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XXXIV.

THE MORPHOLOGIC CHANGES IN THE NOSE AND FACE DUE TO THE DEVELOPMENT OF THE BRAIN.*

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The central nervous system consists of two parts, the encephalon or brain, and the spinal cord. The first indication of the central nervous system is a longitudinal furrow, called the medullary groove, on the dorsal side of the embryo. This groove is gradually converted into a tube by the uniting of its edges, but before the groove is entirely closed its anterior, expanded end presents three swellings known as the primary fore-brain (prosencephalon), mid-brain (mesencephalon), and hind-brain (rhombencephalon), or the anterior, middle and posterior cerebral vesicles.

The primary fore-brain and hind-brain each divide into two parts, and thus five divisions of the brain are formed. From

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before backward these divisions are called the telencephalon (secondary fore-brain); the diencephalon (primary fore-brain); the mesencephalon (mid-brain); the metencephalon or cerebellum; the myelencephalon or medulla oblongata. The cerebral hemispheres of the brain develop from the telencephalon, and the olfactory lobes spring from the base of the cerebral hemispheres.

The peripheral part of the telencephalon is called the mantle or pallium. The relative development and differentiation of this part of the brain is closely related to the mental development of the animal and reaches its highest development in mammals, especially in man.

At first all of the vesicles lie in the same horizontal plane, but in the course of development a cerebral flexure occurs, and the axis of the vesicles is bent downward so that the mid-brain forms the apparent apex of the brain. In fish and amphibia the cerebral flexure is practically obliterated, but it persists in the higher types of vertebrates, especially in mammals. In the latter class, the original relation of the different parts of the brain is still more complicated by the remarkable development of the cerebral hemispheres, which grow forward and backward and gradually cover all the other parts of the brain. Thus, instead of the various portions of the brain being situated one behind the other, they develop so that eventually they lie above one another. This condition attains its greatest perfection in man.

FISH.

In some fish the fore-brain remains undivided, but in most cases it divides and forms the two cerebral hemispheres. Each hemisphere gives off a forward prolongation, which forms the olfactory lobe. In most fish the brain does not fill the cranial cavity, but is separated from the roof of the skull by a gelatinous fluid. The pallium is a simple epithelial structure. (Figs. A, B and C.)

The olfactory organs are sack-like structures situated well forward in the snout, with one or two openings externally. The mucous membrane in the nose is always raised up in more or less complicated folds, in which the olfactory nerve is distributed.

AMPHIBIA.

The brain of amphibia has large cerebral hemispheres and olfactory lobes. The pallium is a little more highly developed than it is in fish. The cerebellum is very small. The brain of the frog shows a somewhat higher degree of development than the brain of the fish. The different parts of the brain are all in about the same plane, and the brain surface is smooth. The olfactory bulbs are fused in the median line. The olfactory cavities each have two openings, the external nostrils or anterior nares, and the posterior nares which open into the anterior part of the mouth.

On account of the changed method of respiration the nasal fossæ become differentiated into an olfactory and a respiratory portion. The turbinates are simple prominences on the floor and side walls of the fossæ. (Fig. D.)

REPTILES.

The brain of reptiles is more highly organized than that of the amphibia. The brain substance shows a distinct differentiation between the gray matter or cortex, derived from the pallium, and the white medulla. The cerebral hemispheres are well developed, but the cerebellum remains comparatively small.

The olfactory lobes are well developed and extend forward toward the nasal fossæ. Their anterior ends are expanded to form the olfactory bulbs, from which the olfactory nerves arise, and are distributed in the mucous membrane of the posterior superior part (olfactory portion) of each nasal fossæ.

In alligators and crocodiles the extent of the nasal fossæ is much increased anteriorly by the growth forward of the facial region, and posteriorly by the formation of the hard palate, which prolongs the nasal fossæ backward under the brain and base of the skull.

Each fossa is divided posteriorly into two superimposed cavities. The superior cavity is the olfactory portion of the nose and is lined with olfactory mucous membrane. The rest of the cavity comprises the respiratory portion of the nose. The turbinates are all rather simple structures. (Figs. E and F.)

In fish and amphibia the nasal fossæ are directly anterior to the brain and on about the same horizontal plane. The

olfactory nerves extend forward from the olfactory lobes into the nasal fossæ, and there is practically no cerebral flexure. In reptiles, the brain extends over the nasal fossæ somewhat, and the cerebral flexure begins to be evident.

BIRDS.

In birds the brain fills the cranial cavity and is shorter, broader and more rounded in form than in reptiles. The cerebellum is comparatively large and has two small lateral lobes and one large median lobe. The median lobe is marked by shallow radiating grooves which extend down into the cerebellum and carry the gray matter with them, thus increasing its extent. The medulla oblongata has a well marked flexure. The cerebral hemispheres are quite large. They extend backward to meet the cerebellum, and forward as far as the extreme posterior part of the nasal fossæ. The olfactory bulbs are extremely small and the nasal fossæ show a moderate degree of development. Each fossa in most birds contains three turbinates. The anterior one is shaped like an inverted T, and is covered with stratified epithelium. The two posterior turbinates are scroll shaped and are covered with olfactory mucous membrane. (Figs. G and H.)

MAMMALS.

In the lowest types of mammals the cerebral hemispheres are relatively small, their surfaces are smooth, and do not extend over the cerebellum. In the higher types the relative development of the hemispheres is remarkable. They extend backward and completely cover the rest of the brain. The surfaces of the hemispheres are divided into numerous complicated convolutions by deep sulci, and in this way the extent of the gray matter is tremendously increased. This development of the cerebral hemispheres reaches its maximum in man.

The optic lobes are relatively small. The olfactory lobes vary considerably in different types of mammals, and their development corresponds with the development of the olfactory organ. In the animals which have an acute sense of smell, with well developed ethmoidal turbinates, the olfactory lobes are relatively large.

This condition is well shown in the dog. The olfactory

lobes are large and form the anterior portion of the cerebrum. The greater part of the anterior boundary of the brain cavity is formed by the cribriform plate of the ethmoidal bone, which is hemispheric in shape and contains numerous perforations through which the branches of the olfactory nerves pass and are distributed over the ethmoidal turbinates.

The marked development of the cerebral hemispheres in the dog carries them forward as well as backward, so that they extend over part of the nasal fossæ anteriorly, and over the cerebellum posteriorly. (Figs. I and J.)

The monkey's brain shows a high degree of development, which is only surpassed by the development of the brain in man. The difference between the human and anthropoid brain is less than the difference between any other two vertebrate groups. (Figs. K and L.)

The human brain, in the course of its development in the embryo and in early life, passes through, in regular order, conditions characteristic of some of the lower vertebrates. The brain of a two-year-old child resembles very closely the brain of an ape. There is, however, a very great difference between an adult human brain and the brain of an ape. In the human brain all of the lobes are relatively much larger, the sulci are deeper and the convolutions are more intricate, and thus the amount of gray matter is tremendously increased.

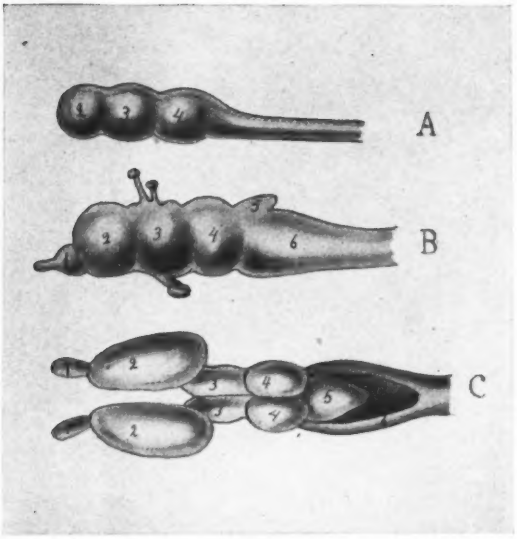
The skull and features of a young ape resemble very closely those of the human fetus, but the adult skulls in each case show very marked differences. The cranial cavity of the ape is small and the jaws are large and protruding, but in man the cranial cavity is actually and relatively very large, while the jaws and face are small and subordinate to the cranium. The principal cause of this difference between the human skull and the skull of an ape is found in the greater development of the human brain, which continues to grow until man reaches adult life, while the brain of an ape reaches its full development very early in life.

The tremendous development of the human brain carries it forward so that it extends over the nasal cavity and changes the facial angle. The nose lies almost directly under the brain, instead of being anterior to it, and the cribriform plate forms the roof of the nose in man, whereas in most other vertebrates it forms the posterior boundary. The olfactory organ

in apes, and especially in man, is a decidedly degenerated structure. The turbinates are small and the olfactory sense is not acute, but this degeneration is more than compensated by the wonderful development of the brain.

By comparing the cranial and facial axes and planes we see that in fishes and amphibia there is no cerebral flexure. The nasal fossæ are directly anterior to the brain and on the same plane. In reptiles and birds the brain, as it develops, extends forward over the nasal fossæ and shows some flexure, and there is a slight facial angle. (Fig. M.)

In mammals, as we ascend the scale, we see a continuous development and increase in the size of the cerebral hemispheres and a marked cerebral flexure. The facial angle increases from 0 to 90°, and the form and position of the nose and face are completely changed.



FIGURES A, B AND C.

DIAGRAMMATIC DRAWINGS OF THE DEVELOPMENT OF THE BRAIN IN A FISH.

Figure A represents the expanded end of the medullary groove, with its three divisions.

2.—Fore-brain or anterior vesicle.
3.—Mid-brain or middle vesicle.
4.—Hind-brain or posterior vesicle.

Figure B shows the second stage in the development of the brain, in which the primary fore-brain and hind-brain have each divided into two parts and the olfactory lobes have formed.

1.—Olfactory lobe.
2.—Telencephalon or cerebrum.
3.—Diencephalon, with the pineal body and the pineal eye above and the pituitary body below.
4.—Mesencephalon, from which the optic lobes arise.
5.—Metencephalon or cerebellum.
6.—Myelencephalon or medulla oblongata.

Figure C.—Dorsal view of the fully developed brain.

1.—Olfactory lobes.
2.—Cerebral hemispheres.
3.—Diencephalon.
4.—Optic lobes.
5.—Cerebellum.
6.—Medulla oblongata.

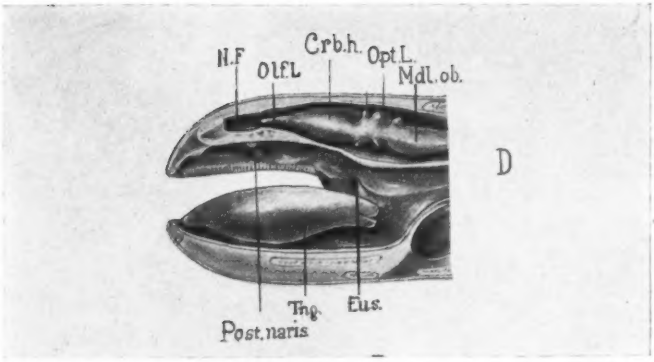


FIGURE D.

LONGITUDINAL SECTION THROUGH A FROG'S HEAD.

N. F.—Nasal fossa, exposed by removing the septum.
Olf. L.—Olfactory lobe of the brain.
Crb. H.—Cerebral hemisphere.
Opt. L.—Optic lobe.
Mdl. Ob.—Medulla oblongata.
Post. naris.—Posterior naris, which opens into the anterior part of the oral cavity.
Tng.—Tongue.
Eus.—Pharyngeal opening of the Eustachian tube.

The different parts of the brain are all in about the same plane. The brain surface is smooth, without convolutions or sulci.

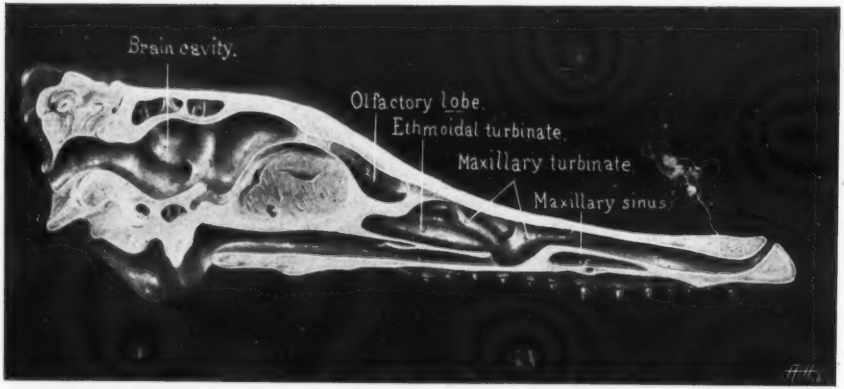


FIGURE E.

LONGITUDINAL SECTION THROUGH THE HEAD OF AN ALLIGATOR.

1.—Brain cavity.
2.—Cavity which contains the olfactory lobe. The olfactory lobe becomes very much constricted at first as it extends forward from the cerebrum, but it expands considerably at its anterior end and divides into numerous branches which pass through the cribriform plate to the olfactory portion of the nasal fossæ.
3.—Ethmoidal turbinate.
4.—Maxillary turbinate.
5.—Maxillary sinus.

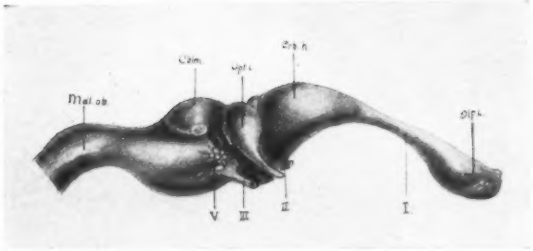


FIGURE F.

THE BRAIN OF AN ALLIGATOR.

Olf. L.—Olfactory lobe. At its extreme anterior part, the severed ends of the olfactory nerve are shown.
Crb. H.—Cerebral hemisphere.
Opt. L.—Optic lobe.
Cblm.—Cerebellum.
Mdl. Ob.—Medulla Oblongata.
I, II, III and V.—Cranial nerves.

The whole brain shows a higher degree of development and the subdivisions are more distinctly marked than they are in the amphiba, but the brain surface does not show any convolutions.

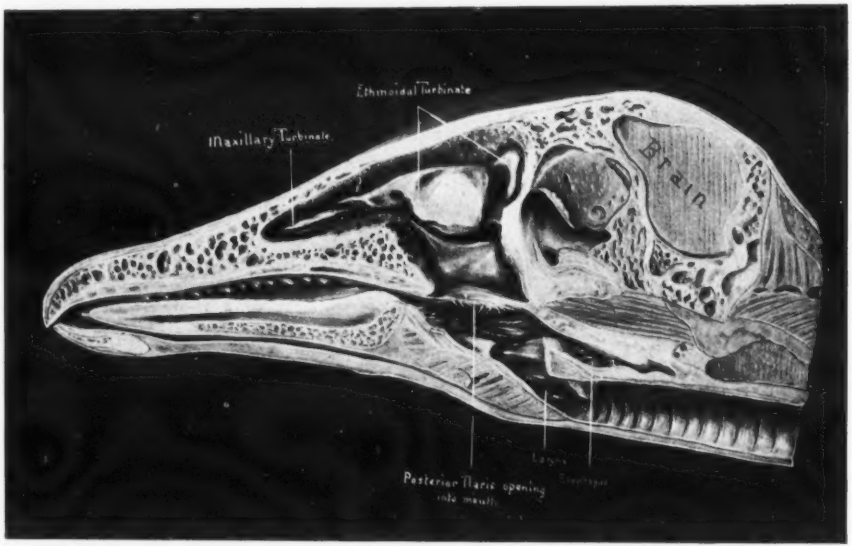


FIGURE G.

LONGITUDINAL SECTION THROUGH THE HEAD OF A GOOSE.

The large air cells in all the bones are characteristic of the bony structure of birds, and make the skeleton lighter. The maxillary turbinate is a T-shaped structure attached to the superior lateral wall of the nasal fossa. In a longitudinal section only the median surface of the two arms of the T can be seen, and the support of this turbinate is entirely covered so that it looks like an irregular projection from the lateral wall. The external opening of the nose is at the anterior end of this turbinate. The anterior ethmoidal turbinate is a double coil. One branch coils upward and makes one complete turn; the other branch coils downward and makes two and one-half turns. The median surface of this turbinate also gives no suggestion of its complicated structure. The posterior ethmoidal turbinate is simply a ridge-like structure and contains air cells.

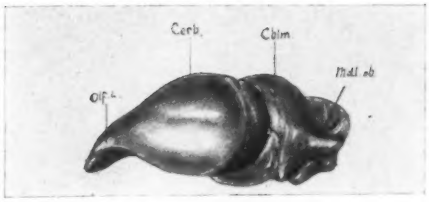


FIGURE H.

BRAIN OF A GOOSE.

Olf. L.—Olfactory Lobe.
Cerb.—Cerebrum.
Cblm.—Cerebellum.
Mdl. Ob.—Medulla oblongata.

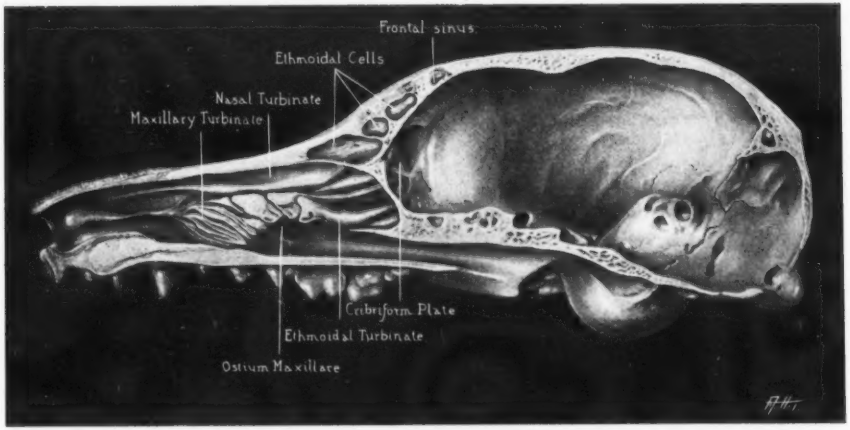


FIGURE I.

SECTION THROUGH THE HEAD OF A DOG.

The maxillary turbinate is a complicated structure with many branches. Its anterior end has a cartilaginous prolongation which extends forward to the extreme anterior part of the nose. In sniffing, this prolongation partially closes the respiratory portion of the nose and directs the inspired air through the olfactory portion. The ethmoidal turbinates are delicate structures with numerous fine divisions. All of the accessory sinuses contain some branches of the ethmoidal turbinates.

FIGURE J.

THE BRAIN OF A DOG.

The cerebral hemispheres show a very high degree of development with well marked convolutions and sulci. The olfactory lobes are relatively large and form the entire anterior portions of the cerebrum. The cerebellum is comparatively small and is partially covered by the cerebrum.

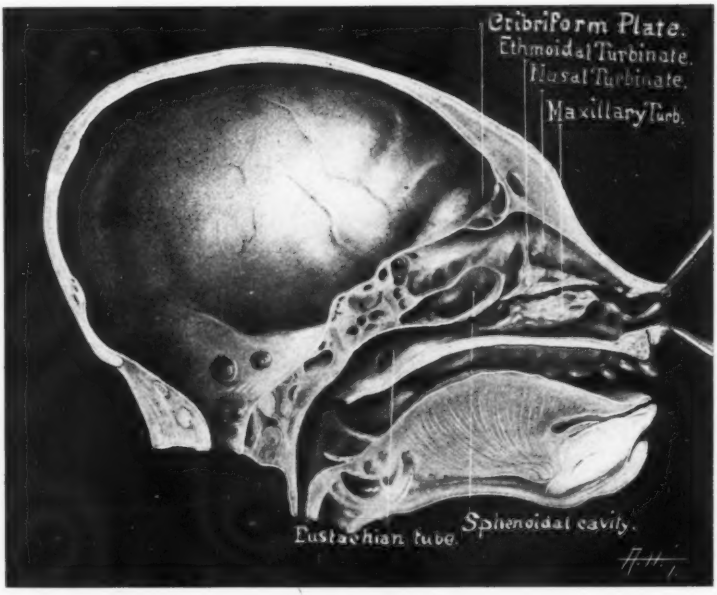


FIGURE K.

BRAIN OF A MONKEY (CEBUS).

The brain cavity extends over the posterior half of the nasal fossæ, and the cribriform plate forms the roof of the nose. The maxillary turbinate has a double coil. There is only one ethmoidal turbinate. The nasal turbinate is rudimentary.

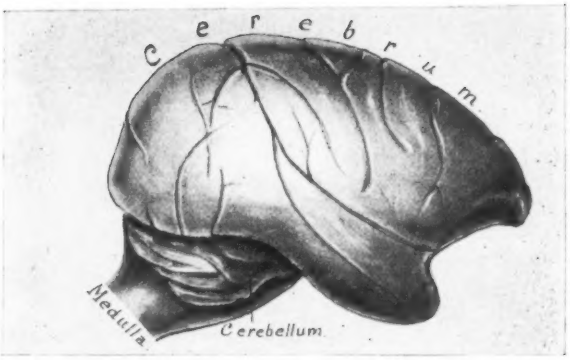


FIGURE L.

BRAIN OF AN ADULT MONKEY (CEBUS).

The cerebrum is large and completely covers the cerebellum. The sulci and convolutions are quite similar to those of the human brain, but less intricate.

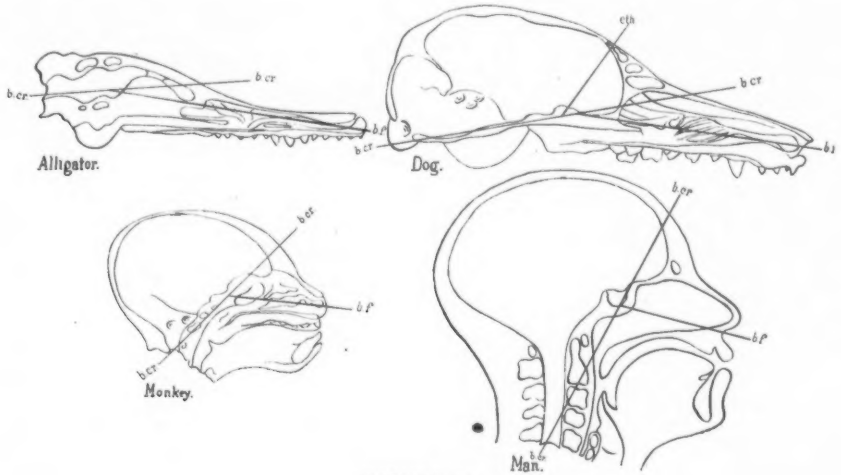


FIGURE M.

b. cr.—Basi-cranial axis.
b. f.—Basi-facial axis.
eth.—Ethmoidal axis.

XXXV.

THE PREVENTION OF DEAFMUTISM.*

By Macleod Yearsley, F. R. C. S.,

London.

The time has surely come when deafmutism should no longer be a specialty within a specialty, but that all otologists, instead of a small majority, should turn their serious attention to its study. It is time that every one of us should possess a practical knowledge not only of the causes which lead to deafmutism, but also of the psychic problems presented by the deafmute and of the methods of dealing with him educationally. The entrancing interest which the study of the deaf child carries with it would arouse every otologist to a sense of the grave duty which falls upon him in the prevention of educational deafness and, armed with a practical knowledge of the subject, he would be in a position to cooperate with the eugenist, the hygienist and the legislator to put into motion such machinery as would diminish the number of deafmutes, congenital and acquired, in the community. Such a movement should be international, and I have, therefore, brought the matter forward at this congress, in the hope that some pronouncement upon it may be made officially by so authoritative a body.

But although a lively sense of his duty to the deaf child requires to be awakened in many otologists, that is not all: the general practitioner must be roused from his slumber also and taught that the same duty exists for him, and that it carries with it more onerous responsibilities even than it does for the specialist, because it is the general practitioner who sees the potential deafmute first and has the opportunity for preventing the establishment of the defect. It is to him that the parents turn for advice and, instead of putting them off with an indefinite promise that the child will "grow out of it" or that he "will hear in seven years," he should be both able and ready to give them practical advice and help, either to save

*Read before the Ninth International Otological Congress, Boston, 1912.

the child's hearing or to educate him if it is past help. Every deaf school contains many scholars who are the victims of lost opportunity.

I have been engaged recently in an inquiry into the causation of educational deafness with special reference to prevention, and in order to arrive at reliable conclusions, I have analyzed 1076 cases personally examined by me in the London County Council Deaf Schools, the school at Fitzroy Square, and the Royal School at Margate, referring also to former statistics obtained at the two latter institutions. The total number of cases analyzed was 2197. For reasons of accuracy I have discarded all doubtful cases, so that the greater number of my analyses has been confined to the 1076 cases which came under my personal observation, whereby I have been able to check the histories and causes given by parents or guardians by physical and functional examination.

Of these 1076 cases, 484, or 44.98 per cent, were born deaf (including 5 cases of congenital aphasia), and 592, or 55.0 per cent, had acquired their defect; and I propose to give a brief summary of the facts elicited by their analysis and to indicate the lessons which are to be learned from them in reference to prevention.

Leaving out the 5 cases of congenital aphasia, the number of born deaf cases were 479. I found that these could be arranged in three groups, those in whom no family history was obtainable, those in which the family history contained no evidence of other deaf births, and those in which there was a distinct family history of congenital deafness, either in the direct line, in collateral branches, or among the brothers and sisters. The first group and the second probably included instances of true sporadic congenital deafness and undetected acquired deafness. I think it may be taken that all cases of deaf-born children fall naturally into one of two primary classes: that of true hereditary deafness, in which the family history shows other deaf-born persons, either in the direct line or in collateral branches, and cases of sporadic congenital deafness in which the family history is deficient in other deaf-born relatives, but may contain instances of other defects, mental or physical. Some of those in the second class quite possibly may be cases in which the deafness was really acquired in early life. Indeed, it is quite common to find that,

in many children believed to be congenitally deaf, the defect was not detected until as late as the second year of life, and may have been due to some postnatal cause which has escaped recognition. No doubt, as our knowledge and methods improve, the real percentage of true sporadic cases will still further be reduced.

Of my 479 deaf-born cases, no family history could be obtained in 148, in 186 there was no family history of congenital deafness, and in the remaining 145, other examples of deaf birth occurred. The first group was of no value for statistical purposes, the second group showed chiefly, as possible etiologic factors, illegitimacy, insanity, chronic alcoholism, and the presence in the family of such other defectives as mental deficients, imbeciles, physical deficients, and epilepsy. There were also eight instances of cousin marriage.

Time will not permit of my going into detailed particulars of the facts elicited by an analysis of group 3, and I can only, therefore, give their salient features. Out of 145 cases, representing 123 families, 30 showed congenital deafness occurring in the direct line, a percentage of 24.3 per cent. Many of these showed deaf birth in collateral branches, and there were, in addition, 26 cases in which this feature only was present, so that, taking both direct and collateral deafness, the 123 families showed deaf birth appearing in either direct line, collateral branches, or both, in 39, or 31.7 per cent. In one family, a man who had a deaf-born sister was the father of 12 children, of whom 6 were born deaf and 3 of these developed retinitis pigmentosa. Lastly, there were 48 families, or 39.02 per cent, in which there was more than one child born deaf or otherwise defective. It is an established fact that, in investigating the part played by heredity in deaf birth, the collateral family tree is of equal or even greater importance than the direct ancestry.

The next point to be taken is the question of consanguinity. Out of 309 families (taking groups 2 and 3), cousin marriages occurred in 22, or 7.08 per cent, a percentage in striking contrast with that of 592 families of acquired deafmutes, in which only 2, or 0.32 per cent, were the offspring of cousins. At Fitzroy Square, out of 137 families, consanguinity occurred in 37, or 27.0 per cent. It must be remembered that this school has had a large proportion of Jewish children among

its pupils and that Jews intermarry more often than do other denominations, so that the high percentage may be thus accounted for. If the two percentages here given be combined, we have 446 families with 59 consanguineous marriages, or 13.25 per cent. The normal proportion of cousin marriages in Great Britain is probably about 1 in 60 or 1 in 70 (1.6 to 1.4 per cent).

Other points to be noted in the family histories of these congenital deafmutes are alcohol and insanity. Both are difficult to obtain reliable information upon, as they are both family peculiarities which are likely to be concealed. I only succeeded in obtaining a history of insanity in 3 families and of alcoholism in 5 out of 309 families (0.97 per cent and 1.6 per cent respectively), statistics which are of no real value, save as showing the difficulties in the way of investigation.

One other point is of great importance in the problem of sporadic congenital deafness, and that is syphilis. I have shown, as also has Castex, that congenital syphilis is a potent cause of acquired educational deafness in children, but very little is known as to its influence upon deaf birth. Baratoux has published some investigations which are very significant, and more recently Mayer has followed on the same lines. The researches of both observers indicate that a certain number of sporadic congenital deafness may be due to syphilis, and I have long held the opinion that this is so. It occurred to me in October last that it might be possible to settle the question by means of the Wassermann reaction, and at the time that this paper is being written I am engaged upon a small investigation on these lines, and I hope that, when this paper is read, I shall have some results to offer to the congress.

Passing now to the acquired cases. These numbered 592. Time will not permit of more than an enumeration of them, their percentage, and the types of deafness found in each. The latter is essential, as it is only by a knowledge of how these various causes bring about educational deafness in children that we can hope to make advances in prophylaxis.

The infective diseases come first, numbering 214, or 34.4 per cent. Of them 156, or nearly 72.9 per cent, belong to the infectious fevers; that is to say, of the whole number of the acquired cases, 26.3 per cent, or more than a quarter, owed their origin to the exanthemata. Taking these in order they were as follows:

Measles. Sixty-eight cases, or 11.48 per cent, the deafness being due to suppuration in 15, middle ear catarrh in 11, middle ear catarrh and suppuration in 1, internal ear disease in 25, and meningitis resulting in internal ear disease in 6.

Scarlet fever. Fifty-eight cases, or 9.79 per cent. Deafness due to suppuration in 42, suppuration with internal ear involvement in 1, middle ear catarrh in 2, middle ear catarrh and suppuration in 1, and internal ear conditions in 12.

Diphtheria. Twelve cases, or 2.02 per cent. Deafness due to suppuration in 6, internal ear disease in 4, and middle ear catarrh in 2.

Pertussis. Seven cases, or 1.18 per cent. Deafness due to suppuration in 3, to internal ear conditions in 4. Banks Raffle (School Hygiene, i. p. 98) has recently pointed out that suppuration may occur during this disease by the forcing of infective material up the eustachian tubes during the spasmodic cough. Deafness may be due also, probably, to intralabyrinthine hemorrhage.

Influenza. Four cases, or 0.67 per cent. Deafness due to internal ear disease in 3, and internal ear disease following convulsions (meningitis?) in 1. It is possible that some of the cases classified as deafness due to middle ear suppuration may have originated in influenza, especially when we consider how often the disease affects the ear. Mygind has pointed out that it is surprising that Wilde is the only author who mentions influenza as a cause of deafmutism, giving it as forming 0.6 per cent of the acquired cases in Ireland in 1851.

German measles. Two cases, or 0.33 per cent. Deafness in both due to internal ear conditions.

Enteric fever. Two cases, or 0.33 per cent, both internal ear in type.

Epidemic cerebrospinal meningitis. One case, or 0.16 per cent. Deafness due to internal ear disease. This fever does not occur with great frequency in England, thanks to the efficiency of the health department. It is, further, more adequately treated than formerly. Were it otherwise, the percentage would probably be higher. It may be noted that Hartmann has given it as responsible for no less than 26.8 per cent of acquired deafmutes in Pommerania-Erfut in 1874-75.

Varicella. One case, or 0.16 per cent, deafness due to middle ear suppuration.

Mumps. A rare cause of deafness, considering the number of children attacked by the disease. One case, or 0.16 per cent, internal ear in character.

Smallpox, once a fertile cause of acquired deafmutism, does not appear in my statistics, although I find it mentioned once in those of the Margate school for 1909. It shows what prophylaxis can accomplish, for there can be no doubt that smallpox has practically disappeared from deafmute statistics since its powers have been shackled by compulsory vaccination.

Leaving the exanthemata, there remain to be considered pneumonia, rheumatic fever, tuberculosis, and congenital syphilis.

Pneumonia was responsible for 16 cases, or 2.7 per cent. In 11 the deafness was due to internal ear disease, in 4 to middle ear suppuration, and in 1 to middle ear catarrh.

Rheumatic fever was given as a cause in two cases, but one of them proved on investigation to be due to middle ear catarrh following adenoids. The other was one of total deafness, due to internal ear trouble, which appeared during the attack of acute rheumatism. The percentage of this disease was, therefore, 0.16.

Tuberculosis. Under this head I have included only such cases as could be proved to be due undoubtedly to tubercle. They were only 2, or 0.33 per cent. In one, the deafness followed tuberculous meningitis, the child suffering total loss of hearing during the attack. The second was a case of tuberculous middle ear disease, which had undergone some fourteen or seventeen operations, and in which the hearing was reduced to one foot for the loud voice. It is possible that tubercle was the original cause in some of the cases of middle ear suppuration, becoming masked later by other infections. It is probable, however, that the small percentage of tuberculous cases in the statistics of deafmutism (varying, according to Mygind, from 0.1 per cent to 1.3 per cent), is due to the fact that in the great majority of cases the child attacked by tuberculosis of the temporal bone dies before school age.

Congenital syphilis furnishes some of the worst examples of acquired deafmutism, cases that are not only deaf, but often seriously blind. This fact does not seem generally recognized, because every cause but the right one is given to account for

its occurrence. One well-known otologist has made the dogmatic and amazing statement that congenital syphilis does not cause deafmutism! My cases number 39, or 6.5 per cent. The causes furnished were "unknown," "fall," "paralysis," "inflammation of the brain," "improper medical attention," "chorea," "whooping cough," "blow," "nerves," "consumption," "tubercle," "chicken pox," "broken leg," and "weakness," and only medical examination revealed the true state of affairs. All the cases were well marked, all had eye complications save one, most had Hutchinson teeth, and, curiously enough, in those which might have been considered as doubtful, Wassermann's reaction was positive. In statistics compiled by laymen in charge of institutions it can hardly be expected that this disease would figure largely, for obvious reasons. Kerr Love mentions 2 cases in 110 acquired deafmutes (1.8 per cent) and Castex found, in the Paris institutions, 18 cases in 719, or 2.5 per cent. My percentage is, therefore, a high one, and I think you will all agree with me that even a small percentage of such cases is a disgrace to a Christian country. Congenital syphilis affords a fine field for prophylaxis, one in which our profession should join hands with the eugenist and the legislator.

In the next group of children, the deafness was due to diseases of the nervous system. The cases numbered 111, or 18.7 per cent. They were distributed thus: Meningitis (excluding epidemic cerebrospinal meningitis and tuberculous meningitis) 65, 19 (18 per cent) of which occurred consecutive to an injury of the head. Forty-five other cases were ascribed to "fits" (26), "convulsions" (12), "brain fever" (1) or "inflammation of the brain" (6), but no evidence was forthcoming by which one could differentiate them from meningitis. One case (0.16 per cent of the total acquired cases) was due to hydrocephalus. Diseases of the brain and meninges play an important part in the causation of educational deafness acquired after birth. I am inclined to the opinion, also, that some of the cases described as born deaf—sporadic congenital cases—really owe their defect to this cause. The statistics quoted by Mygind vary from 11.9 per cent (Ireland, 1881) to 54.5 per cent (Pommerania Erfut), but some of these include epidemic cerebrospinal meningitis. Every case of the 111 caused by diseases of the nervous system showed

internal ear deafness and was probably due to extension from the meninges to the labyrinth.

Our next group contains cases of primary ear disease. It is in this class of acquired deaf case that prophylaxis is particularly possible—nay, easy. The cases comprised number 176, or 29.7 per cent, all, with one exception, being due to middle ear conditions, suppuration or catarrh. The exception was a single case of otosclerosis of early onset, resulting in deafness to an extent sufficient to render the child educationally deaf at the age of eleven. Otosclerosis has not, I believe, figured in deafmute statistics before.

There were 86 cases of middle ear suppuration, or 14.3 per cent. Instances of suppuration due to the infectious fevers are not included. One of them might have originated in middle ear tuberculosis, but the evidence was not sufficiently strong to justify its inclusion under that heading. Forty-three undoubtedly resulted from adenoids, and 2 to other nasal causes. Of the whole 86, 19 were totally and 67 partially deaf. What I wish to emphasize is that at least 45 of the 86, or 52.3 per cent, were due to nasal causes, the vast majority being adenoid cases. That is to say, over half of them were eminently preventable.

The cases of middle ear catarrh are equally interesting and instructive from a prophylactic point of view. Like the congenital syphilitic children, their condition was often ascribed to other causes, such as "teething," "gastroenteritis," "fright," and the like. The deafness was total in 5 cases, partial in 84, the total number being 89, or 15 per cent. Five showed secondary internal ear deafness. Investigation showed that no less than 85 out of the 89 (95.5 per cent) originated in nasal conditions, 80 (89.7 per cent) being due to adenoids. Taking the suppurative and catarrhal cases together, we find that 130 out of 175, or 74.2 per cent, were dependent upon preventable causes, or further, dealing with the whole 592 acquired deafmutes, 21.9 per cent were due to preventable middle ear disease. This conclusion is a somewhat staggering one and is rendered all the more striking when we realize that these figures represent only children whose deafness is so severe as to necessitate the highly specialized educational treatment of the deaf school and that there are numerous others receiving education in ordinary hearing schools who are suf-

fering from lesser degrees of deafness, some of whom are doomed to become much more deaf in adult life. Is not this alone sufficient justification for the remark with which I opened this paper, that not only do otologists require to be awakened to a realization of their duty to the deaf child, but general practitioners educated to that sense?

I now pass to the variety of conditions grouped together under the head of miscellaneous causes. These may be dismissed in a very few words. They number 58, or nearly 9.8 per cent of the acquired cases. Injuries form their great bulk, 50 or 86.2 per cent of them (including 5 cases of concussion of the brain) being traumatic. These do not include injuries which resulted in meningitis. The accidents which gave rise to the deafness in these children included falls or blows on the head, and "run over." All were cases of internal ear deafness, 29 being totally and 21 partially deaf. Injury is included in most statistics as a cause of acquired deafmutism, and the deafness in such cases is probably due to fracture of the base of the skull or to concussion of the labyrinth.

The other miscellaneous causes required to be very carefully investigated. Burns or scalds were said to be responsible in three cases, one of which proved to be really due to middle ear catarrh, under which heading it is included. Two (or 0.33 per cent) remain unaccounted for, unless by the causes given. Both showed internal ear deafness, said to be due to a bad scald at thirteen months, the other to shock following burns, the scars of which were evident. Mygind includes burns among his "doubtful" causes.

Shock or fright was stated as a cause in 9 cases. Investigation showed 1 to be due to middle ear catarrh and 5 probably congenital, under which groups they are included. This left 3, or 0.5 per cent, all of which were deaf from internal ear causes, in one of which the fright to which it was ascribed was followed by prolonged vomiting.

Teething is not uncommonly given as a cause of early deafness. Ten were ascribed to it, but 7 of them did not bear investigation, which showed that 3 were congenital, 2 due to middle ear catarrh and 2 to middle ear suppuration. The remaining 3 gave a history of "fits" during teething and should, probably, be included under the heading of meningitis.

Lightning stroke occurred in 1 case and sunstroke in 3 cases at the Margate school. Hartmann mentions the former, and 31 cases of sunstroke are reported in the American statistics for 1880. Seventeen cases are entered as due to combined causes. They were instances of acquired deafmutism in which it was not possible to come to any reliable decision from the evidence as to which of the two causes given was the one responsible for the condition. They could be divided into middle ear suppurative, middle ear catarrhal, and internal ear cases. Of the former, 1 was due to scarlet fever and typhoid, 1 to scarlet fever and measles, 1 to scarlet fever and diphtheria, 2 to measles and pneumonia. The catarrhal cases were due, 2 to scarlet fever and measles, 1 to measles and influenza, the internal ear cases being due, 2 to scarlet fever and meningitis, 1 to measles and diphtheria, 5 to measles and pneumonia, and 1 to German measles and influenza. They do not call for any comment.

There were 11 doubtful cases, of which 2 proved to be congenital and 2 catarrhal, leaving 7, or 1.1 per cent. Lastly, 10 were originally classified as cause unknown. One of these was found to be an undoubted case of middle ear catarrh, leaving 9, or 1.5 per cent, in which no cause could be ascertained.

Such were the causes of deafmutism found in the 1076 cases analyzed by me personally. In the time left at my disposal I wish to indicate what appear to me the lessons which they convey to us in the prevention of the most serious of educational defects. I will take the congenital cases first. A study of the statistics of deaf marriages in the United States so impressed Graham Bell, the inventor of the telephone, that he wrote a pamphlet On the Formation of a Deaf Variety of the Human Species in America. Those of my cases in which there was a history of true hereditary deafness showed deaf-birth occurring in the direct line, or collaterals, or both, in 31.7 per cent. The summary and conclusions in that valuable work, Marriage of the Deaf in America, published in 1898 by the Volta Bureau, shows the evil results of these unions, results accentuated when deaf marriage has added to it consanguinity. As regards the latter factor, 7.08 per cent of my cases showed the parents to be blood relations against 0.32 per cent of the acquired cases. It is not possible com-

pletely to eradicate congenital deafness, for a certain number of deaf births will always happen, like other sporadic cases of defect. I am of opinion, however, that their number could be materially reduced by the application of eugenic principles. If marriages of the deafborn and of blood relations and the union of alcoholics, syphilitics, and those with a family taint of insanity could be prohibited, these restrictions alone would be of great assistance. At present (in the old world especially) we can only try to educate public opinion, and this is where our profession should help. The doctor has many grave responsibilities on his shoulders, and not the least of them is his duty to the state. In matters like this, he has a great potentiality for good. He could do much to educate public opinion and he should not allow himself to be deterred from doing so.

Besides legislation against undesirable marriages and trying to shape public opinion, however, there is something of a more positive nature which could be done to eliminate congenital deafmutism—sterilization. People shake their heads at the suggestion now, but they shake them more doubtfully than they did, and in a few more generations it will be given practical support. People talk glibly of the "liberty of the subject," forgetting that Burke defined "liberty" as "equal restraint of all," and so long as that definition is ignored, it may be necessary to restrain for the good of the race and for the happiness and physical well-being of unborn generations.

In respect to sporadic cases of deaf birth, attention must be turned to hygiene, especially to the hygiene of motherhood, and to the control of such racial poisons as alcohol, lead, and syphilis.

When we turn to the acquired cases, we are upon surer ground. I need not say very much to an audience of expert otologists. The details I have already given will be sufficient indication as to the direction which our efforts for the reduction of acquired deafmutism should take. I hope they will be discussed and, what is more, I trust that this International Otological Congress will formulate some strong expression of opinion as to the means to put its conclusions into practice.

The prevention of acquired deafmutism embraces a number of factors. It means, for one thing, better care of children generally, better hygiene, better feeding, better clothing,

better surroundings. It means the resolute fighting of many superstitions, such as the dangers of "stopping a discharge," the laissez aller policy embodied in "growing out of" a disease, when more frequently it is the disease that outgrows the child. It means the combating of the mysterious influence of the number seven in the years of a child's life; I am frequently told that nothing had been done to meet a progressive deafness because the patient would "hear when he was seven." I have often wondered how this queer idea originated and whether the amazing English law that the deaf child shall not be compulsorily educated until he is seven years old was founded upon the same absurdity.

Coming more nearly to our specialty, it means better care of the ears during the exanthemata. Until the infectious fevers are shorn of their potency by preventive medicine there must be a ceaseless watch kept for the aural complications by the attendant physician. The practitioner must be educated to watch for deafness or ear pain and to interfere promptly himself or urge the necessity of immediate expert advice. He must also know how to deal with the throat and nose complications which lead to this development. This would save numbers of ears in scarlet fever, measles, and diphtheria, which, as I have shown, are responsible for the bulk of the exanthematous acquired deafmute cases. Mumps we have seen to be a rare cause; when it appears it is a sure and certain one, unless dealt with very promptly. As regards the reduction of the exanthematous fevers themselves, I can see no reason why they should not one day be made as impotent as smallpox, which has almost disappeared by compulsory vaccination, in spite of the malign efforts of anti-vaccination cranks.

The deafness arising during the progress of congenital syphilis also requires prompt and energetic measures. Ordinary specific treatment is of no avail, and salvarsan requires to be employed with circumspection at present. Some cases become deaf whilst actually under specific treatment for ocular complications. Hence, possibly, the finding of "improper medical treatment" put forward as a cause. Promptly met by pilocarpin, or better, by repeated blistering, it will sometimes be kept down. What is really required, however, is to strike at the root of the disease by making syphilis notifiable.

Discarding cant and hypocrisy, we should set to work to stamp it out as an infective and preventable disease by honest, reasonable, and consistent scientific legislation.

We have seen that the vast majority of the primary ear diseases causing acquired deafmutism is eminently preventable. The profession in bulk has not yet reached to the serious recognition of the evils of adenoids and nasal diseases in infancy, although it is on the high road thereto. We specialists know them well, but the majority of the profession is not sufficiently alive to them. Adenoids must be prevented, or, when present, treated more seriously and efficiently. It is not enough to remove the bulk of them, but Rosenmüller's fossæ must be cleared of them consistently. Barraud, of Lausanne, has recently pointed out that the improper artificial feeding of infants is a potent cause of adenoids, and the past two or three years has seen much done, especially in the United States, in the work of the prophylaxis of the oro- and nasopharynx. The time to treat these cases is before the otitis has developed, and here we must go back to the infant. Of the 592 acquired deafmutes analyzed, 130 out of 175 primary ear cases were preventable. I think the matter would not be immoderately stated if we take half (107 that is) of the 214 infective disease cases as similarly preventable. This would make 237, or 40 per cent, preventable of all the acquired cases, and this is really quite a modest computation. This needs no comment, it is pregnant with meaning.

Lastly, I would plead for a better and more systematic teaching of the principles of otology in our medical schools. Instead of being the Cinderella of the specialties in our curricula, we should not rest until we can insure that no student receives a qualification to practice until he is well grounded in a knowledge of the causes of ear disease and their prevention.

XXXVI.

EPITHELIAL GRAFTING AS A MEANS OF EFFECTING THE SURE AND RAPID HEALING OF THE CAVITY LEFT BY THE COMPLETE MASTOID OPERATION.*

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SYLLABUS.

The argument. The advantages of grafting and the effect on hearing. Alernatives to grafting: Baracz's plastic, the blood clot dressing, and the use of Beck's bismuth vaselin. Grafting in labyrinthine operations. History of grafting. Best time for grafting. Technic. Concluding remarks.

THE ARGUMENT.

Two conditions are essential to the success of operation for the cure of chronic otorrhea: First, all disease must be removed; second, the large bone wound must be made to heal from the bottom. We may say at once and deliberately that, in the great majority of the cases, all the disease can be removed without injury to important structures. Greater difficulty has been experienced in fulfilling the second condition. Systematic tamponing, either through the unsutured mastoid wound, or through the enlarged meatus, has been employed for this purpose. All who have carried this out are aware how painful it is to many patients and how it wearies both the surgeon and the patient. Whiting,[1] in his work published in 1906, speaks of the severity of the pain inflicted by dragging out the gauze packing, and of the weary weeks of frequent dressings and irksome details which the intelligent care of mastoid cases requires. Politzer[2] says: "The duration of the after treatment varies from six weeks up to nine months and over. The mean duration is between three and four months." Grunert[3] says: "In exceptional cases cicatrization proceeds so smoothly that the entire cavity is healed in from

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four to six weeks; on an average cicatrization is complete in three to four months. There are, however, specially unfavorable cases in which, either as a result of constitutional peculiarities or of particularly unfavorable local conditions, this period of time becomes considerably extended." Passow,[4] in a thoughtful paper, gives statistics from various clinics showing that the average duration of treatment by the tamponing method varied from four to ten weeks, in the most favorable instances, up to thirty-six weeks and over. Respecting the most favorable figures quoted, he, however, says: "Though Hartmann, in the most recent edition of his textbook, when referring to the radical operation, remarks laconically that the healing takes place in from four to ten weeks, this can really only be taken cum grano salis." Most striking, however, are the figures which he gives respecting the cases operated on in his own clinic at Heidelberg. Of forty-four recovered cases, eighteen were operated on by different methods, but all without Thiersch grafting. In these the average duration of the after-treatment was twenty weeks. The other twenty-six cases were operated on by a method, described in the paper, of which Thiersch grafting forms a part. In these the average duration of treatment was eleven weeks. One healed in three and a half weeks, but the majority took over three months to heal.

A visit which I paid some years ago to the otologic clinics of Körner, Lucae, Trautmann, Jansen, Schwartze and Grunert, Barth, Politzer, and Bezold, in which I saw hundreds of cases in various stages of treatment by the tamponing method, gave me the impression that the above quoted statements give rather too favorable a view of the results of that treatment. I should say that in many of the cases treatment was continued for six or seven months. I became, therefore, more than ever confirmed in the opinion that the method I described in 1900[5] presents great advantages over tamponing alone and over the methods of grafting practiced before that date by myself and others.[6] In almost all my cases the bone wound becomes dry in from two to three weeks. When grafting is carried out at the close of the bone operation the tympano-antral cavities are epithelialized immediately, and at the end of ten days it often happens that the further treatment no longer calls for special skill. I never obtained such results

without grafting; and I may add that I had a wide experience of mastoid surgery before anyone even proposed to shorten the healing process by grafting. In general surgery skin grafting is carried out in order to hasten the cicatrization of a wound having a large superficial area, since a large wound to which living epithelial cells are applied heals more quickly than one which has to be entirely covered by ingrowth of epithelial cells from the edges. The wound left after the complete mastoid operation is not only of large superficial extent, but its floor is in great part raw bone, a tissue which heals much more slowly than does soft tissue, and is, moreover, much more susceptible of reinfection during the process of healing. The surface of the wound is not flat, but it is so shaped that granulations, if allowed to become exuberant, may cross the cavity and shut off spaces which remain unhealed and which so become a fertile cause of persistent discharge and so of failure of the operation. When I visited his clinic, Jansen, in doing the complete mastoid operation, applied immediately several small grafts, completely sutured the mastoid flap, and treated the case by tamponing through the enlarged meatus. He objected to grafting the tympanum on account of its numerous recesses, which he considered made the proper application of the graft impossible, and also because the graft might throw a veil over the two windows and so produce loss of hearing. This fear is not confirmed by experience. The sole object of the grafting operation is to expedite the healing of the wound. To contend that there is any great difference in the final result is an error, but the more rapid the healing the better for the patient in general, and for the hearing in particular. The result of grafting by the method I now use has been, in my hands, so satisfactory in many hundreds of cases that I can strongly recommend it.

The advantages gained by grafting the mastoid cavity are:

1. Rapid healing of the entire wound, bone, cartilage, and soft parts, on ordinary surgical principles.

2. Immediate protection of the raw bone surface by a layer of living epithelium and, in consequence, elimination of the pain and discomfort otherwise incidental to treatment by tamponing and of the liability to reinfection of the bone.

3. Considerable shortening of the time during which specially skilled attendance is necessary. Two weeks after the

grafting operation the bone granulations are all covered by visible living epithelium, and the rest of the attendance may safely be left to the family doctor.

4. Improvement in hearing. When the graft has been cut very thin and successfully applied, healing takes place at once under the best conditions. The graft, except its inner layer of living cells, separates by aseptic molecular necrosis. The fenestræ become covered by the thinnest possible layer of tissue, and consequently the hearing is generally very good, and is certainly much better than when granulation has long been present over the fenestræ.

These advantages are almost invariably obtained when all bone disease has been removed and the raw bone surface has been covered with a single graft, cut sufficiently thin, and accurately applied. If these conditions have not been fulfilled the method described by me has not been carried out.

THE HEARING AFTER THE COMPLETE MASTOID OPERATION.

Patients naturally always wish to know whether they will hear better or worse after the operation than before, but this question cannot be answered with certainty. In the absence of disease of the labyrinth, the condition of the parts around the fenestræ is the main factor influencing the final result as to hearing. Several observers (for example, Politzer, Heine, and Grunert) have given statistics on the subject which show that on an average the hearing is improved in rather less than half the cases, unaltered in about a third, and made worse in the remainder.

Several years ago I tested the hearing in a number of consecutive private cases. The operations were made from one to seven years after operation. In every case operation had been done for long standing otorrhea, with more or less bone disease. The ages of the patients ranged from 14 to 50 years. In all these cases my method of grafting was carried out. About 75 per cent of these patients showed remarkably good hearing as the result of the operation. I propose at a future time to publish further details about them. In children the hearing after the complete mastoid operation, combined with my method of grafting, is usually very good. They may suffer no educational disadvantage and their deafness may even pass unnoticed while they are at school.

ALTERNATIVES TO GRAFTING.

The only alternative to grafting I have so far mentioned is tamponing, but we have also the method of Baracz, the "blood clot dressing," Beck's bismuth vaselin paste method, and the use of scarlet red.

In 1900, Baracz of Lemberg[7] published a method he had devised of covering the bone with a flap of skin taken from the neck. This plan has the disadvantage of leaving a scar in the neck, but it can be rapidly carried out and requires less delicate manipulation than does the grafting operation which I recommend. Such a flap as Baracz makes should never be placed over the inner wall of the tympanum, where the thinnest of Thiersch grafts alone are suitable. Those who apply thick slices of tissue over the fenestræ must not be surprised if the hearing of their patients is not improved. I have no personal experience of this method.

THE "BLOOD CLOT DRESSING."

After the mastoid operation a cavity is left in the bone, the walls of which cannot be approximated; if the skin be sutured over this a "dead space" is left which fills with clot. Surgeons have always regarded such "dead spots" with considerable distrust, and it was until quite recently generally taught that the presence of blood clot in such spaces was harmful. Only a few years ago a drainage tube was considered indispensable, even for a clean wound made through healthy tissues, lest blood and serum should accumulate in the wound and prevent healing. In the fifth edition of the late Greig Smith's Abdominal Surgery, published in 1896, the objection is raised to the method, now so generally in use, of suturing the abdominal wall in layers, that "such a suture leaves a series of gaps to be filled with clot, between each layer at the line of junction."[8]

Further observation showed that this fear of blood clot in a wound was groundless. So far from exercising a deleterious influence, it is an important factor in the process of healing, since the serum from fresh blood clot possesses bactericidal powers, and the fibrin of the clot forms a delicate scaffolding upon which the new fibrous tissue is built up. In the time of our forefathers it was a common practice, often

attended with the most happy results, to allow the wound in a case of compound fracture to become occluded with dried blood, but the reasons why many of these cases did well were not then appreciated. In 1886, Schede[9] published a paper "On the healing of wounds under a moist blood scab," in which he showed how important a part blood clot plays in the obliteration of "dead spaces." In 1891, Halsted,[10] to whom we are indebted for many improvements in surgical practice, called attention to the subject and pointed out that not only should clean wounds be as a rule entirely closed, but that suppurating wounds might, with certain precautions, be similarly treated. Among the cases he mentions in support of his views are several instances of bony cavities. In commenting on these he says: "A bone cavity should never be stuffed. The granulations should be encouraged to grow as luxuriantly as possible. Whether the blood clot melts away or not, the bone cavity should be bridged over by skin or by protective gutta percha tissue. The granulations must be most carefully protected from insult. They should rarely, if ever, be irrigated. In other words, a bone cavity which has lost its blood clot should be treated as if it still possessed it." The method advocated in Halsted's paper for dealing with "dead spaces," which has been termed the "blood clot dressing," is deliberately to allow the space to become filled with fresh blood, to close it over with skin, if skin be available, or if not to cover the opening with gutta percha tissue, to place over all a protective dressing and to let it alone. By this means it is claimed that rapid healing may be obtained in many cases which, if treated by the open method, would heal much more slowly and with greater deformity.

The success obtained in dealing with other bone cavities by this method has induced several surgeons to use it in mastoid cases. I have, indeed, myself applied it successfully in acute cases. If the "blood clot dressing" is to be employed, no antiseptic must be used, as it is essential to success to have a wound surface of uninjured living tissue.

When I was in Copenhagen last year I found that Professor Mygind adopted the "blood clot dressing" as his ordinary method of treating cases of the complete mastoid operation. A week or ten days after the first operation he throws forward the pinna and curettes the cavity. He then allows the cavity

to fill with fresh blood, sutures the flap in place and applies a protective dressing. I do not consider, however, that the results of this method can be compared with the rapid healing obtained by epithelial grafting, and I may add that the results I have obtained from the "blood clot dressing" in acute cases compare unfavorably with those obtained by the more common surgical methods. Moreover, after the complete mastoid operation the object in view is not to fill the cavity with a mass of cicatricial tissue, but to epithelialize the surface of the cavity as rapidly as possible.

BECK'S BISMUTH PASTE.

I have found this method most useful in promoting the healing of cavities elsewhere in the body, and I have used it both as a substitute for and as an adjunct to grafting in mastoid cases. It outlines the cavity, sterilizes its surface, checks exuberant granulations, and does not interfere with the growth of epithelium. It obviates the necessity for skillful tamponing. I make the injection once a week.

SCARLET RED.

I have not observed any great advantage from the employment of scarlet red ointment (*Annals of Surgery*, Vol. 51, 1910) in the cavity left by the mastoid operation, possibly because my experience of its use in these circumstances is small. In complicated cases (extradural abscess, sinus thrombosis, etc.) in which the superficial wound is often large and is designedly left widely open, there is no doubt in my mind that the epithelialization of the superficial wound and of the edges of the mastoid cavity is much hastened by the use of this ointment. Last month I used the scarlet red ointment with great success in the convalescence of two patients, from extradural abscess and cerebellar abscess respectively. The scarlet red ointment should be applied every other day; on alternate days some other simple dressing.

GRAFTING IN LABYRINTHINE OPERATIONS.

If I may look forward a little, I think that epithelial grafting will be found useful in hastening the healing of wounds which are now so commonly made in the petrous for the cure

of suppurative or nonsuppurative conditions. Recent operations for tinnitus and vertigo have been attended with a fair measure of success, and there is some hope that deafness due to obstruction of the fenestræ may some day yield to operation.

Many patients have complete tympanic deafness without any, or with but very slight, evidence of disease of the nervous apparatus of hearing. In these cases no sound waves can penetrate the bony box of the internal ear through the normal channels, and it seems possible that aerial conduction might be restored by making an artificial opening in the capsule of the cochlea.

THE OPERATION.

The complete mastoid operation is done, the posterior wall of the osseous meatus and the annulus tympanicus being especially well cut down so as to get a good view of the inner wall of the tympanum. The mucous membrane of the tympanum and antrum is not disturbed. The stapes is then carefully removed and the little wedge shaped portion of bone between the fenestræ is cut away with a gouge of corresponding width; the stroke should be gentle and should be directed from above downwards and forwards. The greatest care should be taken that the cutting edge of the gouge does not cross the vestibule and impinge upon its inner wall; a little clear fluid escapes. The opening is immediately covered over by an epithelial graft just large enough to overlap its margins. No further escape of fluid occurs, and healing takes place rapidly and satisfactorily with the usual dressings. An opening so made leads into the vestibule and into both scalæ of the cochlea. Possibly it might be better to open the cochlea directly and avoid opening the vestibule. In a case I published many years ago,[11] suppuration had extended into the semicircular canals, and in removing these the vestibule was widely opened; it was not the seat of suppuration, and clear fluid was seen to escape from it. A graft was placed over the opening into the vestibule. The result was remarkable—not only was the vertigo cured, but the hearing was restored. The patient was a woman, 54 years of age, who had had left otorrhea since childhood. At the time of operation there was absolute deafness of the left ear, and the patient said she had been deaf in

that ear all her life. The graft over the vestibule was absorbed and replaced by a living membrane which could be identified by a probe passed through a speculum. I have always thought that sound waves, in this case, passed through the membrane which had formed over the aperture in the vestibule made at the operation, and not through the fenestræ. This is another route by which aerial conduction may possibly be restored.

During the last fifteen years I have operated on the labyrinth for deafness in a few selected cases, but only in one with any success. I shall try again.

I should like to refer to Jansen's method of epithelial grafting in acute cases, but time does not permit.

HISTORY.

For some years before adopting epithelial grafting systematically, I had from time to time applied grafts, either at the time of doing the bone operation or at varying periods afterwards, sometimes through the unclosed retroauricular wound, sometimes through the meatus. I do not claim priority in the matter of the application of Thiersch grafting, a procedure well known in general surgery, to the mastoid operation. Several German, and probably enough other, surgeons had made use of epithelial grafts in mastoid operations considerably before my method was published; but I believe that I was the first to advocate the systematic adoption of epithelial grafting for the immediate healing of the bone wound, and that this should be combined with closure of the wound by suture. The account of this method was published in January, 1900.[12]

A description of Thiersch's method of transplanting large epithelial grafts is given in a paper published by Urban of Leipzig in 1886.[13] The first mention of Thiersch grafting as a part of the complete mastoid operation is, so far as I am aware, in a paper by Siebenmann, published in 1893.[14] Siebenmann recommended the application of epithelial grafts (2 cm. by 5 to 7 cm.) two or three weeks after the bone operation.

Reinhard of Duisburg in 1894[15] endorsed Siebenmann's recommendation, and in 1898[16] wrote more fully on the subject. He had tried immediate grafting in 1893, but soon gave it up on account of the unsuccessful results, due, he says, partly to

faulty technic and partly to selection of unsuitable cases. In the 1898 paper he advised grafting at the first, second, or third dressing after curetting the granulations under an anesthetic. He held that it was never possible to say with any certainty that all disease had been removed, and that cases in which it was advisable to close the mastoid wound were exceptional. He concludes the paper by saying that he had no doubt that epithelial grafting, notwithstanding its often considerable technical difficulties, would become more and more appreciated, whereby the practice of the radical operation, so beneficent in its results, would no longer be restricted through the fear of a prolonged aftertreatment.

In 1897, Alfred Denker, then of Hagen, in Westphalia,[17] now Geheimrath Professor in Halle, speaking at the German Otological Congress in a discussion following a paper by Nolentius on the mastoid operation, suggested grafting by the Thiersch method in order to expedite cicatrization of the open retroauricular wound. In 1899[18] he read a paper on the subject, and advised grafting from the second to the fourth week. He cut a large graft which, he said, could, with a little practice, easily be done quickly and without an anesthetic; this he divided with scissors into several portions, which he placed in position by means of Jansen's spatula; any graft over he replaced on the thigh. He left the graft exposed to the air, only placing a small strip of sterile gauze on the floor and in the lower angle of the operation cavity. The operation region was then protected by means of a sort of thimble, which was kept in place by bandages. Several well-known surgeons joined in the discussion on this paper. Siebenmann said that he gave ethyl bromid, or more rarely employed "suggestion narcosis," to curette the granulations and cut the graft, and that he placed the graft in position after the patient had recovered from the narcosis. He advised that the whole surface should be grafted.

Passow said that he found grafting less successful after the burr had been used than when the whole operation had been done with gouge and mallet. In a letter written to me in April, 1904, Professor Passow said: "I employ Thiersch grafts about eight days after the operation, but only if the discharge is small in amount. I have seen no advantage from grafting at the time of operation."

Rudolf Panse (at the 1899 discussion) expressed doubt as to the value of grafting, and attributed the good results to the second curetting. In a letter written to me in April, 1904, he said: "I consider Thiersch grafting (a) as dangerous if the flaps cover any diseased bone, (b) as unnecessary, for after the removal of all disease healing takes place in six weeks." Scheibe (at the 1899 discussion) said that he grafted at the first dressing. It is evident from the mention of Jan- on the granulations. Jansen advised grafting immediately or sen's spatula, and from Jansen's own remarks at this dis- cussion, that he was one of the pioneers in this field. Stoddart Barr[19] has quite recently described a method of inserting an epithelial graft through the meatus; he cuts the graft under local anesthesia.

WHEN THE GRAFTING SHOULD BE DONE.

The graft may in a few selected cases, with advantage, be applied immediately on the conclusion of the operation for removing the disease. To conclude the whole matter at one sitting is certainly most desirable, but as Celsus[20] said: "Danger is apt to attend attempts to effect a cure too quickly and too pleasantly." The operation cavity is never surgically clean, and is often very foul, and I think the ultimate result will most often be better if the grafting is postponed for some days and the cavity again curetted. In a few complicated or specially foul cases it may be better to leave most of the wound unsutured in the interval, but, ordinarily, I close the mastoid wound entirely and apply a light dressing. I do not plug the operation cavity, but have it irrigated twice daily through the meatus with a mild antiseptic solution. When a sufficient interval, usually seven or eight days, has elapsed, the wound is reopened and the surface of the tympanoantral cavity prepared anew by removing clots and granulation tissue. No antiseptic, which might destroy living cells, may be employed during the grafting stage. In the presence of intracranial complications the grafting operation is, of course, postponed until the issue of these dangerous conditions is known. There is no doubt in my mind that the second curetting of the wound is in certain bacterial infections a great advantage. In general surgery such treatment is often employed, and I have found it of conspicuous utility after the mastoid operation when the infection of the soft tissues tends to persist.

TECHNIC.

Epithelial grafting is carried out in precisely the same way, whether it follows immediately upon the conclusion of the bone operation or after an interval.

All bleeding having been arrested, the operation cavity is finally cleansed with hydrogen peroxid and warm sterile saline solution. The skin of the thigh or arm having been previously prepared, a large epithelial graft, as thin as possible, is cut with a hollow-ground razor moistened with warm sterile saline solution. The inner aspect of the upper part of the thigh yields as a rule an excellent graft, because here a flat surface of sufficient extent is readily obtained by stretching the skin. A convex surface or a concave surface, such as that over Hunter's canal, gives a graft of varying thickness. The cutting of a graft sufficiently large and thin requires practice and dexterity, and this is, I believe, the main difficulty in the universal acceptance of the method. The skin varies much in different individuals. A coarse skin does not yield a very thin graft; the finer the skin, the thinner and more perfect is the graft. The skin of children yields beautiful grafts.

It is quite easy to cover the root of the antrum and the attic and the inner wall of the antrum with one graft, but it is more difficult to apply the graft to the inner wall of the tympanum proprium. The graft is best carried to the wound spread out on a microscope section lifter. These lifters are made of steel, nickel plated, and are of various sizes. The front border of the lifter is placed against the outer or superficial edge of the anterior wall of the cavity in the bone made by the operation. The margin of the graft is now coaxed from off the lifter onto this superficial edge, and also above onto the adjoining superficial edge of the roof of the cavity, just below the linea temporalis. The upper and anterior margins of the graft thus placed are held in position by a probe. The section lifter is gradually withdrawn, and the lower and posterior edges of the graft drop against the posterior and lower boundaries of the operation cavity; the graft thus bridges over the operation cavity. The air and blood which separate the graft from the inner wall of the tympanoantral cavity are removed by a little skillful manipulation, but chiefly by suction through a pipette, insinuated beneath the edge of

the graft. The graft then clings closely to the raw bone surface, adapting itself accurately to its eminences and depressions. The graft is now held flat by the atmospheric pressure against (1) the anterior wall of the cavity, formed internally by the anterior boundary of the tympanum and attic, and externally by the anterior wall of the enlarged osseous meatus; (2) the anterior part of the roof of the cavity, formed by the tegmen tympani and the superior wall of the enlarged osseous meatus; (3) the inner walls of the attic and tympanum; (4) the tegmen antri; (5) the tuberosity formed by the horizontal semicircular canal and the fallopian canal, and (6) the inner wall of the antrum.

A little bleeding is apt to come from the neighborhood of the eustachian tube, and the pipette may have to be used more than once before the graft lies satisfactorily against the tympanic wall. If the tegmen has been removed the graft lies against the dura. It is undesirable to graft the posterior and lower part of a very large mastoid cavity, for this will make the permanent cavity unnecessarily large. Moreover, this part will heal rapidly enough by the approximation of the raw inner surface of the mastoid flap to the granulating bone surface. The main effort should be directed to covering the tegmen, the inner wall of the attic, tympanum, and antrum with epithelium so that these cavities, which have for years been the site and harbor of disease, may be immediately epidermized.

The graft is held in place against the bone by the application of small, dry, sterile cotton wool mops or a strip of narrow ribbon gauze dusted with a nonirritating antiseptic powder such as aristol. If the little plugs are used the first is placed in the lower part of the tympanum and often extends into the upper part of the eustachian tube, the next is placed against the upper part of the inner wall of the tympanum and attic, and the next against the inner boundary of the aditus ad antrum. About seven plugs are usually employed. A piece of sterilized gauze is then placed over all to prevent displacement. The little cotton wool mops are held in angular forceps and pushed home by a steel probe or stopper, the smooth head of which prevents any chance of the graft clinging to the stopper as it is removed. One or two grafts are placed on the outer boundary of the operation cavity—namely, the

inner surface of the mastoid flap—corresponding in extent to the tympanum, attic, and antrum.

It is a good plan to arrange a portion of these external grafts so as to cover the raw edge of the posterior margin of the meatus, for here the cartilage is exposed without a complete skin covering. The edges of these grafts are thus inveigled through the meatus and appear on the skin surface of the concha. The mastoid flap is replaced and sutured with fine silkworm gut. A tiny gauze mop is placed in the meatus to support the grafts against the raw posterior edge of the meatus and a dry sterilized dressing is applied. The external dressing should be renewed daily. The plugs are removed with forceps from the third to the sixth day. This procedure is painless, as the raw bone is protected by the graft. The subsequent treatment consists in irrigation with hydrogen peroxid and rectified spirit twice a day, or in dry gauze tamponing as often as may be thought desirable. When the dead part of the graft has come away, the cavity, on careful inspection, is seen to have a smooth pink surface, which is in fact a surface of epithelium and no longer a raw surface of granulations. In the course of a few days the color of the surface changes from pink to pearly gray, showing unmistakably that the grafting has been successful. The last place to heal is a cartilage granulation, which usually presents just inside and behind the lower margin of the meatus. If tamponing is employed, sterilized ribbon gauze, half or one inch in width, is well adapted to the purpose. It may be impregnated with a mild astringent, such as aluminium acetate. Iodoform gauze should not be used, as it promotes flabby and exuberant granulation.

Victor Hugo[21] said: "Every new offspring of science presents this double aspect, monstrosity as fetus, wonder as germ." The history of all great operations is the same. Pathology was for long in advance of surgery, and a germ from which, after long years, a scientific procedure was evolved, often at first gave rise to a monstrosity of an operation. The mastoid operation is no exception. It commenced with crude and imperfect efforts and at length reached a stage free from danger and death, and in our day is conducted upon "a plan leading direct to the end desired."[22] We must not on this account rest satisfied with our present methods and results.

"While anything remains to be accomplished," observed a great statesman, "nothing is done." The mastoid operation will be still further improved. "Look one step onward and secure that step," said Paracelsus.[23] In the attempt to follow that advice this paper has been written.

ADDENDUM.

I.

MR. SYDNEY SCOTT, aural surgeon to St. Bartholomew's Hospital, has sent me an interesting letter on the subject of mastoid and labyrinthine grafting:

"June 24th, 1912.

"I am very much pleased to tell you my views and experience of skin grafting in connection with the radical mastoid operation, small though this experience is, compared with what yours must be. The first mastoid case which I grafted some five years ago was one of malignant disease, in which there was an unusually large excavation of the temporal bone. During the last three or four years I have made a rule of grafting every case as a part of the regular procedure of the radical mastoid operation; not at a subsequent stage, but at the same time as the mastoid operation. This immediate grafting undoubtedly hastens healing in a remarkable way, so that instead of having to keep patients under close daily observation for four to six weeks, one can allow them to leave after the second week, and subsequently they require very little attention, apart from the precautions against proliferating granulations from some spot which may have escaped the graft. The economic result, as regards hospital patients, has been that many more can be passed through the few beds which are allotted for mastoid surgery, since the adoption of immediate grafts, than was possible in the days when no grafts were applied.

"I take it that the object of grafting is to obtain a more rapid healing of the surface of the cavity than is possible without grafts. Incidentally, grafting prevents contraction of the cavity walls, and of stenosis, which used sometimes to be noticed before grafting was adopted. Generally speaking, I have not found age any contraindication It is sometimes convenient to take the graft from another person, in the case of an infant, or when there is much wasting.

"I employ the immediate graft, whether suppuration exists or not. In cases of extradural abscess complicating chronic otitis, for which one performs the radical mastoid operation, I employ a graft, and close the postaural incision just as one does in uncomplicated cases. Whether the whole of the graft adheres or only a portion of it, I am convinced of the saving of considerable time in the healing process.

"Again, in cases of postoperative suppuration where the immediate graft was applied, the graft is not necessarily destroyed by the infective inflammation; even in quite unpromising looking cases (which are fortunately not common), a considerable portion of the graft has been found to have adhered as the reaction subsides.

"Coming to labyrinth cases, I have no reason for not grafting over a simple fistula of the external semicircular canal when the labyrinth is functionating, but when a fistula of the labyrinth is accompanied by acute infective labyrinthitis, it is, in my opinion, not permissible to graft without first securing adequate drainage of the vestibule; and even then it is questionable whether a graft over the opened labyrinth is advisable. In such cases, and generally in operations on the labyrinth, when relatively deep seated pockets have been made into the labyrinthine capsule and are bridged across by the facial nerve (as, for instance, in extirpation by the double route of vestibulotomy), it is better to limit the graft to the meatomastoid portion of the cavity, leaving the labyrinthine cavities to become filled by reparative material, and this soon becomes epithelialized if one can prevent proliferation beyond the general level of the grafted surface.

"Nor should I employ a graft over the labyrinth if the internal auditory meatus has been opened, as indicated by the escape of cerebrospinal fluid. Believing this escape of fluid acts as a safeguard and tends to prevent leptomeningitis, I have no objection to applying a graft to the meatomastoid part of the cavity in such cases.

"To discuss the important question of the effect of grafting on the acuity of hearing would necessitate looking up a long series of cases in order to give exact details. I must be content with saying that I avoid applying grafts to the inner tympanic wall in cases in which useful hearing has survived the prolonged suppuration, but when great deafness exists,

and is unlikely to be relieved by the operation, I do not hesitate to graft over the promontory and fenestræ."

II.

"My colleague, Mr. Marriage, aural surgeon to St. Thomas' Hospital, has sent me the following account, which is eminently practical, of his experience of epithelial grafting:

"'June 28, 1912.

"'In reply to your inquiries re the grafting of mastoids, it has been my practice for the last five years, in all cases of chronic mastoid disease, to graft the cavity at the time of the removal of the disease, but in cases of acute mastoid disease, where the radical operation is necessary, I do the grafting about a week or ten days after the primary operation.

"'The method I have adopted in the chronic cases is to thoroughly remove all trace of disease, then clean out the cavity with hydrogen peroxid (20 vols.) and apply one large graft, which should be very thin. The graft is got into position by sucking out the air beneath in the manner which was first recommended by you, and is kept in its place by one long sterilized plug of plain gauze one-half inch in width, which is covered with aristol powder to keep it from sticking to the graft; the end of the plug is brought out of the meatus and the graft then wrapped over the plug and also brought out of the meatus so as to cover the cut edge of the meatal wall and form a complete covering for the entire cavity.

"'At first I used to leave the plug in for seven days, but I now find that five days is sufficient for the graft to become adherent. After the removal of the plug the ear is syringed out daily with a weak solution of hydrogen peroxid and a small piece of gauze put in to absorb the discharge; this is carried on for six or seven days, and afterwards patients are given hydrogen peroxid (10 vols.) to drop into the ear twice daily. The primary grafting, in addition to saving the patients the trials of a second operation, considerably shortens their stay in hospital, as usually they are able to leave at the end of about twelve days; it also saves them the pain of constant firm plugging, and is much less troublesome to the surgeon, for after they leave hospital it is only necessary for them to come up once a week for inspection and for the cauterization of any small granulating areas which may be pres-

ent. The majority of cases are healed by the end of six weeks, the last place to heal being usually the cut edge of the posterior wall of the meatus.

"'Even when it has been necessary to expose the dura of the middle and posterior fossæ, I apply a primary graft, as it grows on the dura extremely well. I have also grafted in the same way a dozen cases in which there was a bony fistula in the external semicircular canal, with excellent results.

"'One case, a lady of 35, who had had otorrhea for over twenty years, was sent to me as possibly a case of cerebellar abscess, as she had violent headache and very marked vertigo, and was quite unable to walk straight. I diagnosed a fistula of the semicircular canal, and at the operation found that she had a large cholesteatoma which had eaten away the arch of the horizontal semicircular canal; I cleared away all the disease and applied one large graft in the usual way, and within six weeks the cavity was completely healed. About two months later I happened to meet her at a dance, and was able to prove for myself that she could dance through a long waltz without any trace of giddiness. The operation was done four years ago and there has been no recurrence of the disease or of the vertigo.'"

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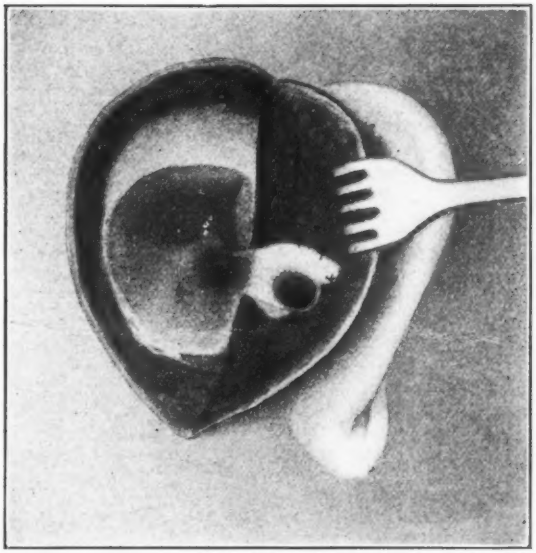

FIGURE I.

The complete mastoid operation. The meatal flaps above and below, freed from cartilage, are shown stitched to the raw surface of the mastoid flap.

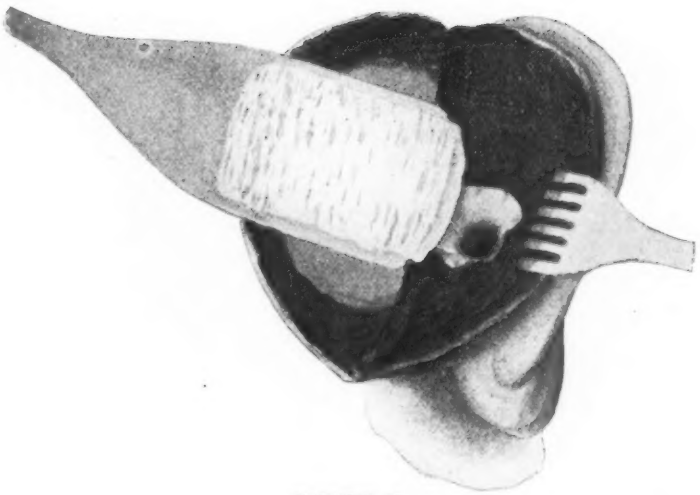

FIGURE II.

Shows the graft on a lifter placed over the bone cavity. The sutures of the meatal flaps have been removed. The bone operation had been performed seven days previously.

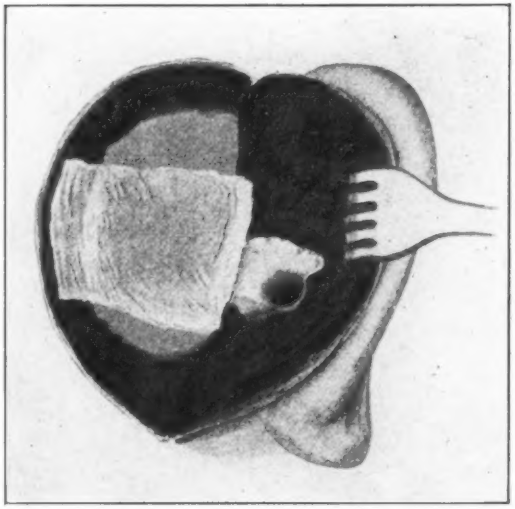

FIGURE III.

Shows the graft lying over the bone cavity, the lifter having been taken away.

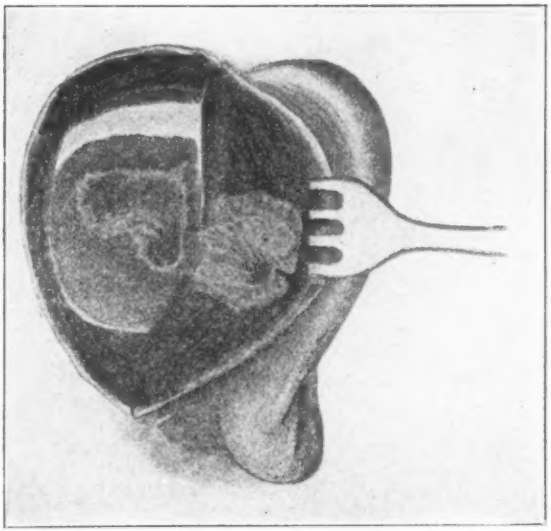

FIGURE IV.

Shows the graft lying against the inner walls of the tympano-antral cavities and slightly overlapping their vertical boundaries. Note that the graft is not allowed to pass below the lower margin of the antrum. On the outer boundary (inner surface of the mastoid flap) two smaller grafts have been applied. Note that a portion of each graft has been coaxed round the edges of the external meatus, the posterior half of which is a raw cut edge.

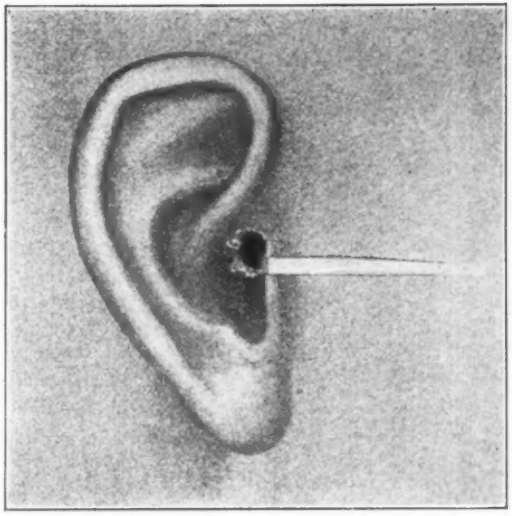

FIGURE V.

Shows portions of the two grafts on the inner surface of the mastoid flap appearing on the external aspect of the meatus. They are thus caused to cover the raw cut edge which forms the posterior half of the new meatus.

NOTE.—In these figures the flap formed by the posterior wall of the membranous meatus is shown turned upwards. Some surgeons turn this flap downwards. In either case there remains a raw cut edge which forms the posterior half of the new meatus. I often use a Körner flap cut very wide. When this method is employed there is no raw cut edge to the posterior boundary of the meatus, and the inner aspect of the outer boundary of the operative cavity does not require the application of a graft.

XXXVII.

THE AFTER-TREATMENT OF MASTOID OPERATIONS.*

Including an Analysis of Results in 260 Radical and 100 Conservative (Heath) Operations.

By J. Walker Wood, L. R. C. P., L. R. C. S., Edin.,

London.

PRELIMINARY REMARKS.

Any mastoid operation is but the commencement of a course of treatment the aim of which is the arrest of discharge, the preservation of life, or of hearing, although before the operation, imminent danger may have rendered the question of hearing quite a secondary consideration. Satisfactory repair after the operation can usually only be obtained by careful and painstaking management, for no matter how well the operation has been performed if the after-treatment is neglected or carried out unintelligently, failure is bound to result. Before operation the surgeon would be wise if he warned his patient that the after-treatment might take a considerable time, as failure to do this has occasionally led to the misapprehension on the part of the patient that once the operation was performed all discharge would immediately cease. If he is asked when the discharge is likely to cease, a guarded prognosis should be given, though it is is usually arrested within three months. A case may be perfectly dry within a month, though such a result is exceptional. Endeavors have been made by various aural surgeons to shorten this period of repair by manifold methods, some of which I will describe later.

The improvement in hearing which accompanies repair is often considerable, though after the radical mastoid operation hearing is never again perfect. Patients are sometimes disappointed after this operation at finding when the discharge has ceased that they are more deaf than they were immediately

*Read before the Ninth International Otological Congress, Boston, 1912.

after the operation. This should be explained to them. During the healing the membrane around the stapes is in a more or less moist condition, allowing of free movement of that bone in the oval window; when, however, the membrane has become dry, or when scar tissue has formed around it and contracted, the movement of the bone is somewhat impeded, and deafness results.

PREOPERATIVE DETAILS.

The intimate relationship of the mouth, nose and throat with the ear must never be forgotten. In children, unless in cases of urgency, the throat and nasopharynx should be examined for adenoids and tonsils, and if found, their removal is essential, either before further operative procedures on the ear or, in urgent cases, at the time of the operation. In adult patients the teeth should be examined previous to the operation by a competent dental surgeon and the mouth placed as far as possible in a healthy condition. As is well known, nasal obstruction is a common factor in the causation of deafness and aural catarrh, a blocked nose and a deaf ear on the same side being often found. The commonest forms of nasal obstruction met are those due to the various forms of rhinitis, septal deviations and nasal polypi. A case has been reported of accessory sinus disease (antrum) causing suppuration in the ear. Free nasal respiration is essential to healing following the mastoid operation by lessening the tendency to nasal, eustachian, and aural catarrh. In female patients the hygiene of the hair and the scalp is of importance; the night previous to the operation the head should be washed and dressed with some mild antiseptic lotion, and just before the operation is commenced and while the patient is under the anesthetic the auricle and meatus should be cleansed by washing with soft soap and water, followed by an evaporating spirit lotion. Mr. Stuart-Low, an aural surgeon very successful with his mastoid cases, soaks the meatus well at this time with pure turpentine. Only by strict attention to details of this kind can one hope to obtain good results in mastoid surgery.

OPERATIVE DETAILS.

Before describing in detail the after-treatment of the various mastoid operations, it will be well to consider one or

two points which, although they apply to the performance of the operation itself, and especially the radical operation, have a bearing on the subsequent course of the case.

1. The mastoid antrum may vary considerably in its position in different individuals. It may be higher up or lower down in the mastoid process. It may be near or far from the surface and may also vary in size. Whatever its position is, it must be completely opened, so that the cavity is rendered freely accessible from the meatus. Should there be any cells or sinuses in direct communication with the main cavity, they, and the antrum, should be incorporated into one large cavity, for all pockets and recesses must be avoided. To gain access to the antrum as much of the posterior bony wall of the meatus as is necessary, and of the facial ridge as is safe, should be removed, along with the outer attic wall. At the same time the arched floor of the meatus requires leveling, for only in this way can tympanic discharges have free exit.

2. Instruments used. The use of the burr increases very considerably the amount of discharge following operation. This is, I believe, due to a superficial necrosis resulting from the grinding and surface destruction of the bone. It is obvious also that a certain amount of bone dust must be ground by the burr into the fine cells of the mastoid process, and as this cannot all be removed by irrigation, each of these minute particles forms a tiny sequestrum, which has to be absorbed by nature. In addition to the superficial necrosis induced by the burr, some damage to living tissues may be caused by the heat generated in the forcible use of this instrument. Those who have used the burr much, or have seen it used, will appreciate my meaning. Cases in which the burr has been extensively used, usually remain "wet" (i. e., discharging) longer than others, and the discharge is more apt to be foul and excessive. The burr is of service if one wishes to leave a bony cavity with beautifully smooth walls. Such a cavity is not desirable, for the smoother the walls the slower is the growth of granulations.

3. Removal of fragments, ossicles, etc. It is essential that all fragments of bone which have been separated by the gouge should be immediately removed. For the rapid performance of mastoid operation two assistants are necessary; one for sponging, the other to remove bone chips. I can remember

some instances in which a prolonged postoperative discharge, out of proportion to the size of the wound, has caused me to suspect the presence of such a fragment, and an investigation has led to its discovery in the lower part of the tympanic cavity (the cellar). These fragments may after a time become enveloped by granulations, and suspicions of their presence may only arise when a sinus is observed in an otherwise healthy cavity. A bent probe is required in order to locate these fragments. Neither should the ossicles (malleus and incus) be forgotten, their removal in the radical mastoid operation being made certain, otherwise they may act as "foreign bodies" and prevent healing.

4. Foreign bodies in the wound. Strands of gauze or wool are liable to be left behind after an operation and thus act as foreign bodies. The short gauze strips used by some surgeons for mopping during operations often have frayed ends, and shreds are liable to adhere to the rough bone surface, remaining in the wound and preventing healing. As a substitute I can recommend the wool mops made in various sizes, according to the stages of the operation, and slightly moistened to aid absorption, which were introduced by Mr. Charles Heath, for their use ensures a drier wound and thus the saving of much valuable time. If strips are preferred for sponging they should have the running edge selvaged, and be not more than six inches in length.

5. The most desirable treatment of the tympanic orifice of eustachian tube is a point which has given rise to various expressions of opinion. Shall we curette around the orifice or shall we leave it alone? If left, reinfection of the mastoid wound may take place by septic organisms from the throat; for it is well known that throat organisms are frequently found in aural discharges. Yet, even if we curette, there is no certainty that the eustachian orifice will close. Probably a middle course is best, and I would suggest that in the radical operation, if the resulting cavity is a large one, and a considerable amount of bone disease is found, combined with the history of many years' suppuration, it is best to leave the eustachian tube alone. For this reason, should the discharge continue after operation, as it might well do under the circumstances mentioned, then a natural drain is provided for any secretion, and the patient is relieved of the discomfort of a continuous meatal discharge. On the other

hand, should conditions appear favorable to the early cessation of discharge after operation with a dry ear, then I would recommend curetting the orifice of the tube, though even then, as stated, there is no certainty of cutting off the connection of the tympanum with the nasopharynx. Sometimes in spite of every effort of the surgeon the opening refuses to close. This may be due to, (1) inability to keep the part at rest, i. e., during the act of swallowing air is pumped into the tympanomeatal cavity, disturbing any attempt at healing which may have occurred; (2) the character of the mucous membrane which alters abruptly at the tympanic orifice of the tube; (3) caries around the bony orifice I have observed more than one case where a continuance of the discharge was due to caries around this tympanic opening, and it is probable that had the curette been used, this might have been prevented. If this orifice is still open at the end of three or four months, it may be touched with the galvanocautery or a small skin graft applied. It has been suggested (Heine) that when a portion of the drum membrane is left it should be utilized as a graft to cover the tympanic orifice of the tube. The wonderful regenerative powers of the membrana tympani are well known. I have never seen fragments of drum used in this way, but I have noticed that when a portion of the drum has been left after the radical operation, it has taken on active growth and ultimately formed a diaphragm which appeared to aid hearing very considerably. I have notes of five such cases.

6. Formation of a flap. The chief objects of cutting a flap from the meatus are to shorten the period of healing and to provide room for the inspection of the cavity. Every surgeon appears to have his own method of cutting and anchoring this flap and each will probably consider his own the best. Whatever method is adopted a large meatus must be provided for and the flap so fixed as to aid, and not retard, drainage and inspection. It should also be gently pressed against the raw bone surface, in order to prevent effused fluids from accumulating beneath it, which might push it forward, and thus occlude the meatus. One great advantage of a tube in the meatus is its elastic effect in pressing the flap against the bone. A flap is not absolutely necessary; in several cases I have seen the flaps cut off, and in others they have sloughed off, and their loss only entailed a prolonged convalescence.

CONSIDERATION OF AFTER-TREATMENT.

In the description of the aftertreatment of mastoid operations the following operative procedures will have to be considered: Wilde's incision, the cortical mastoid operation, Heath's conservative mastoid operation, and the radical mastoid operation.

Before describing the postoperative treatment it should be stated that whatever method is adopted, strict attention should be paid to personal asepsis. After protection of the head and shoulders of the patient with a mackintosh, these parts should in all cases be covered with sterilized towels wrung out of a weak carbolic lotion. The surgeon's hands should be carefully cleansed, as must also those of the nurse in charge of the dressings. Instruments ought to be boiled. If forceps are used in dressing a case, separate pairs should be provided for cleansing the wound and for applying the dressings. Plain aseptic gauze has been found to be more satisfactory than the medicated gauzes so much in use.

1. Wilde's incision in these days is usually a temporary expedient practiced in order to gain relief until a more extensive operation can be performed on the mastoid antrum itself. After the incision the part is treated on the lines of an open abscess, by fomentations, with or without a small plug of gauze or a tube to assist the drainage by keeping the opening patent. A fomentation should not be put on at the time of the operation, but at an interval of twelve hours afterwards. The dressing should be changed as often as the case requires.

2. The cortical mastoid operation (Schwartze) has also largely fallen into disuse since Mr. Charles Heath introduced his conservative mastoid operation. In early acute cases the cortical method is often satisfactory, but has many disadvantages when compared with the Heath operation. In the Schwartze operation the antrum is fully opened along with any diseased mastoid cells, but the posterior wall of the bony meatus, the membrane and the ossicles are left untouched and usually unseen. The postauricular is kept open and the cavity firmly plugged from behind with ribbon gauze. The original dressing is changed twenty-four hours after the operation. The bandage and the dressing often require to be

loosened with hydrogen peroxid, the plug can then be removed and the wound gently irrigated with warm boric lotion or other fluid, it is then dried and replugged with ribbon gauze.

If the condition of the wound appears to be satisfactory, dressing on alternate days is sufficient, but should the discharge become foul, or there be much pain, with or without a temperature, then daily dressings are necessary. A slight elevation of temperature is general after the cortical operation. It occurs usually within forty-eight hours of the operation and rarely exceeds 102° F., gradually falling to normal in three or four days' time. An interesting study of postoperative temperatures has been made by Harris,[22] to whose writings I would refer those who desire to study them. At each dressing the wound should be inspected with a strong light, with the view of discovering the cause of any trouble. Any discharge accumulating in the meatus requires removal, and the passage from the antrum (the aditus) to the tympanic cavity should, if possible, be kept open as long as any discharge from the tympanum continues. This cannot always be done. To keep the aditus free, Heath's aditus canula is of service; its use will be more fully explained when describing his conservative operation. A small rubber drainage tube may occasionally be used with advantage, and, if anything, results in quicker healing than when the wound is packed. Excessive growth of surface granulations should be checked by the use of bluestone or other astringent. The patient should be kept in bed from five to seven days and may be allowed out at the end of a fortnight. The duration of healing is usually from four to six weeks. A depressed and ugly scar often follows this operation, and occasionally a discharging sinus is left.

3. The Heath conservative mastoid operation. (1) Before considering the after-treatment of this frequently performed operation it is necessary to describe a few of its important details, especially those which have a bearing on the subsequent treatment: (1) The skin incision is made right in the angle where the skin covering the mastoid is reflected on to the cartilage of the ear. (2) Access to the antrum is obtained by removal of practically the whole of the posterior wall of the bony meatus, including the suprameatal spine, right down to within one-fourth inch of the tympanic ring. The arched

floor of the bony meatus is also leveled, thus ensuring freer drainage from the tympanum and antrum. (3) Formation of a flap (in detail). The special double edged flap knife designed by Mr. Heath is passed into the cartilaginous meatus and pushed through the roof just external to the temporal muscle, thus not to injure it. It is then carried outwards for about one-fourth inch, giving space for a pair of dissecting forceps to be inserted in order to hold it open, and the knife is then withdrawn. The incision being made to gape by the assistant, the knife is reentered from above into the external end of this incision and made to cut obliquely downwards, backwards and outwards, reaching the outer end of the floor of the meatus. A long and fine pair of bent scissors are used to cut the inner part of the flap. This flap is then separated at its inner end and laid down on the floor of the enlarged meatus, where it is fixed by a catgut suture to the pericranial flap which was prepared early in the operation for the purpose. Mr. Heath claims for this flap: (a) that it covers the floor of the meatal cavity, which, being the part unseen during the after-treatment, should be well protected; (b) turning the flap down insures that it will not prevent inspection of the antrum, which should be in view, during the aftertreatment; (c) discharges gravitate to the bottom, which is covered with a skin surface, and here, if it were uncovered, the discharge would interfere with healing and cause unhealthy granulations. 4. Any granulations blocking the aditus, and all the mucous membrane of that area and the antrum, are removed, and blasts of air are blown through the tympanum by the aid of the aditus canula. Two small and well fitting wool pledgets soaked in iodoform emulsion* are placed within the cavities, one lying against the membrana tympani, the other in the tiny open antrum, their object being to absorb the plastic lymph which is thrown out and which, if left, usually breaks down within a week in a septic cavity, thereby adding to the discharge. The external auditory meatus is enlarged by cutting from within that passage right through the crus of the helix to admit later a large drainage tube. This tube is next inserted,† it is slit above and below at its inner end, and

*Iodoform in equal parts glycerin and boric acid lotion.

†Mr. Heath says he used a rubber drainage tube for his first mastoid operation in 1890, and has never given it up. He adopted it on account of the pain caused by plugging.

also cut obliquely externally, its apex pointing backwards, to prevent pressure upon the tragus. The object of these inner slits being, in the first place, to avoid pressure, and in the second, to allow discharges to find their way into the tube. The length of the tube requires to be varied, according to the depth of the meatus. The postauricular incision is entirely closed with black silkworm gut sutures. The wound is dressed with wet cyanid gauze, covered with waterproof tissue and bandaged. The following day the stitches are all removed and the dressing alone is changed. The tube is not touched. On the second day after the operation the tube is taken out, a smaller one is usually required, the pledgets of wool are also removed. The wound is cleaned with dry wool (no water or lotions being used), blasts of air are blown through the aditus with the canula and bag, and the patient is also instructed to inflate the ears by the method of Valsalva. Any discharge blown from the tympanic cavity through the perforation is removed with dry mops. Fresh pledgets of wool soaked in iodoform emulsion and the tube are inserted lastly, and a clean external dressing is applied. This method of treatment is practically painless and the hearing at this early stage will usually be found to show some improvement. The dressing as described is carried out daily, and the discharge will now be found to come principally from the antrum, the eustachian tube being adequate to carry off the diminished tympanic secretions. The aditus canula is only required for a few days, as it is desirable that the tympanum should become shut off from the antrum, by the formation of a plug of granulation tissue in the open aditus. At each subsequent dressing the patient is made to "blow through" (Valsalva's method).

For the first week or two daily dressings are required, but as the perforation heals, and the discharge from the antrum becomes less, dressing on alternate days will suffice. The plug in the meatus may at this stage be advantageously soaked in absolute alcohol to prevent the formation of granulations, the antral plug still being soaked in the emulsion as there granulations are required.* The antral plug should be gradually

*Granulations are not wanted in the meatus, but they are in the antrum.

reduced in size as the cavity fills up. The tympanic plug may usually be dispensed with at the end of three to four weeks.

A word regarding tubes in general. In the majority of cases a tube one-half to five-eighths inch in diameter will be found of sufficient size. It is necessary to have a large assortment of tubes graduated in thirty-seconds of an inch, from five-eighths to one-fourth inch or less. The function of the tube is to prevent contraction of the meatus and allow of free drainage from the antrum and tympanum—the secreting areas. It should never cause any pain or ulceration of the walls of the meatus. If found to be uncomfortable, it must at once be changed for a smaller one, cut in a similar manner. On no account should the tube be permanently removed until all discharge has ceased, the perforation closed and epithelialization of the cavity completed.

In a few patients the tube in the meatus is not tolerated for long, then a cylindrical plug of tightly rolled one-inch ribbon gauze can be used, and it should be of sufficient size to fill the meatus and prevent contraction, yet without causing undue pressure.

It is unnecessary to keep the ear bandaged for more than a week; a piece of wool tucked into the concha serves the double purpose of absorbing any slight discharge there may be and hiding the tube.

The perforation may heal within five days, but has been known to take five months when very large, the difference in duration depending on the vascularity of the part and the extent of the destruction.

RESULTS.

It must be remembered that the conservative operation was designed by Mr. Heath primarily to save the hearing. This it has done in hundreds of cases. It has been urged against this operation that it was only suitable for acute cases or cases that might get well by meatal treatment. This idea is controverted by a consideration of the following eight cases, most of whom had had a discharging ear for years and been treated by various and many methods of intratympanic medication. In all the perforation healed after the conservative operation, even in one case where there had been a discharge for forty years, a result impossible of attainment by the instillation of drops or syringing. Without a single exception the hearing

has vastly improved in them all. Indeed so great was the improvement in two cases with bilateral middle ear suppuration that the patients returned, one of them from abroad, to have the second ear put right. I have seen a considerable number of these operations performed both at the Central London Throat Hospital and since I have been associated with Mr. Heath, but I have never yet seen a fatal termination to this operation, although several operations have been undertaken for intracranial complications.

Hearing is restored, discharge arrested and all danger of cranial complications reduced to a minimum. The operation itself is a perfectly safe one. There is no fear of facial paralysis. The scar is invisible. What more can be desired?

During the last two years I have dressed over one hundred of these cases, healing was completed in from four to eight weeks, the average period being six weeks. In only 5 per cent did the radical operation become necessary.

In some cases, it is true, tinnitus has followed the operation, but this has always proved amenable to treatment. I should like to mention a few of the "after effects" of the conservative operation, all of which can be prevented if the aftertreatment is carried out with sufficient skill and knowledge.

CALCAREOUS DEGENERATION.

Calcareous degeneration of the drum is occasionally observed to follow healing. It does not apear to affect the hearing power to any great extent. Should it be associated with fibrous adhesions between the drum and ossicles and inner tympanic wall, it may then cause some impairment of audition.

TINNITUS.

Tinnitus occurs during two different stages of the healing period. It is sometimes observed while the drum is in the vascular state which accompanies repair and appears to be due to increased intralabyrinthine pressure caused by the swollen tympanic mucous membrane. Usually it is only a temporary condition, gradually disappearing as the tympanic structures return to their normal condition. In character it is low pitched and intermittent, but always more noticeable after exertion or any condition which tends to raise blood pressure. No treat-

ment is necessary. The tinnitus which may develop when repair is complete and the ear dry is a much more intractable form. It is also low pitched in character, but more continuous, and caused by fixation of the stapes in the oval window by adhesions, which also block the round window, resulting from the inflammation. Variations in blood pressure during cardiac systole and diastole are transmitted to the fluids within the labyrinth and affect the intralabyrinthine pressure. The membranes of the round and oval windows may be looked upon as safety valves (in addition to their sound conducting functions), which allow these variations in pressure within the labyrinth to take place. Should these windows become blocked then the tension of the whole labyrinthine system is raised and minute stimuli are able to irritate the delicate organ of Corti. The treatment which I have found to be most successful in relieving the noise is frequent inflation of the middle ear through the eustachian catheter. No doubt this acts by stretching the adhesions.

ADHESIONS WITHIN THE TYMPANUM.

Inflammation of a joint always results in more or less stiffness and disability. The intratympanic joints are no exception. Adhesions are liable to form around the malleus, the incus and the stapes, limiting their movements, hampering the action of the tympanic muscles, and preventing the transmission of sound. Deafness, of a greater or less degree, is an accompaniment. The scar of the perforation may, unless care is taken, become adherent to the inner wall of the tympanum. Such a result, although not desirable, is quite compatible with excellent hearing. Much can be done to prevent the formation of these adhesions. At each dressing the tympanum should be freed of all discharges, the patient inflating the ear by Valsalva's method, by this means blowing secretions through the perforation into the meatus where it can be removed with cotton wool mops. The inflation must be kept up until the air passes freely. This also prevents the perforation becoming adherent. Should adhesions have formed, much can be done to stretch them by means of the rarefacteur and inflation through the eustachian catheter. In this class of case I believe fibrolysin, as recommended by Gay French for deafness due to postsuppurative catarrh of the middle ear,

would be valuable. French states, "the earlier the case is treated after the cessation of discharge and formation of scar tissue, the better the prognosis."[18]

PERMANENT DRY PERFORATION.

Some drum membranes are abnormally deficient in their blood supply. Should they become perforated by suppurative disease of the middle ear, they may heal when suppuration ceases after the performance of a conservative operation. A dry perforation, however, may be associated with normal hearing. If it is thought desirable to close a perforation, one may be able to induce closure by applying to the edges of the perforation well diluted blistering fluid, as recommended by Mr. Chas. Heath.[27]

CONTINUANCE OF DISCHARGE.

Continuance of discharge after the Heath operation is rare. The aural surgeon may undertake the conservative operation with the full knowledge that he cannot eradicate all the disease within the ear, but trusts that nature when assisted may complete his efforts. Nature may fail to do so. Suppuration sometimes occurs in what has been a patient's only hearing ear, the other being useless from disease or injury; it is then that risks have to be taken. Supposing the perforation does not heal after operation, the life of the patient is no longer in danger, the dangerous area (the antrum) being obliterated, hearing is not rendered worse, most likely it is improved, and now the eustachian tube is able to deal effectively with the tympanic secretions. Rarely does the radical operation become necessary.

CASES.

No. 1. Miss B., aged 50. Chronic suppuration of the middle ear. Duration, forty years. Followed scarlet fever. L. ear, large perforation involving posterosuperior and inferior quadrants. R. ear, double perforation separated by narrow strip of drum attached to handle of malleus, perforations in both inferior quadrants. Conservative operation, left ear, July, 1907, undertaken for urgent symptoms. Perforation closed in six months. Conservative operation right ear, July, 1909. Perforation closed in five months. Hearing now:

L., 30/50; R., 35/50. Able to follow ordinary conversation.

No. 2. Master S., aged 15. Chronic suppuration of the middle ear—right. Followed scarlet fever. Duration, seven years. Medium sized perforation of posterior quadrants of membrane. Conservative operation, November 9, 1906. Perforation closed in nine days. Hearing is now perfect, 48/50. (See B. M. J., July 13, 1907.)

No. 3. Miss R., aged 35. Chronic suppuration of the middle ear—right. Duration, nine years. Kidney shaped perforation below and behind umbo. Attacks of pain and giddiness, "tympanic dam." Conservative operation, February 3, 1910. Perforation closed in two months Hearing before operation, watch on contact only; can now hear it at 28 inches. Able to follow ordinary conversation.

No. 4. Mr. F., aged 35. Chronic suppuration of the middle ear—right. Duration, twenty-seven years. Large perforation involving practically the whole of the tense membrane. Right ear useless from nonsuppurative catarrh. Hearing became so bad that he gave up his occupation as a clerk. Conservative operation, April, 1908. Three weeks later hearing had improved so much was able to return to work. Perforation closed in five months. Present condition, watch 30/50. Hears ordinary conversation quite well.

No. 5. Miss R., aged 40. Chronic suppuration of the middle ear—right. Duration, seven years. Small perforation in the attic, attic disease. Conservative operation, June, 1909. Perforation closed in two months. Hearing present condition, watch 35/50. Practically perfect.

No. 6. Mr. O., aged 33. Acute suppuration of the middle ear. Duration, six days. Small round perforation, high up, deficient drainage. Headaches and pain. Paracentesis failed to give relief. Conservative operation, April, 1910. Perforation closed in one month. Before operation did not hear watch on firm contact, now hears it at three feet.

No. 7. Miss C., aged 25. Chronic suppuration of the middle ear from childhood. Followed scarlet fever. Medium sized perforation below umbo. Inner tympanic wall granular. Radical operation, left ear, in 1903. Conservative operation, right ear, July 19, 1910. Perforation healed in two months. Almost stone deaf before operation. Present condition, watch 40/50.

No. 8. Miss T. S., aged 33. Acute suppuration of the middle ear. Duration, six weeks. Medium sized perforation above and behind umbo. Pain, headache and mastoid tenderness. Conservative operation, October, 1910. Perforation closed in three months. Hearing, present condition 30/50. Good for ordinary conversation.

THE RADICAL MASTOID OPERATION (STACKE).

In considering the after-treatment of this important operation, I have endeavored to classify the different methods in general use: (1) Methods with a tube. (2) Methods without a tube; (a) dry, (b) wet, (c) plugging. (3) Various methods.

I. METHODS WITH A TUBE.

(a) (Jakins) The first dressing should be done about the third to the fifth day; when in young children, general anesthetic may be necessary. It is unwise to leave a dressing longer; it should be remembered that the mastoid cavity after operation is not aseptic. Should the external dressing of gauze and the wool be stiff with dried blood and discharge, it may be softened by soaking with hydrogen peroxid. The tube when removed should first be washed and then sterilized by boiling. The wound itself is gently mopped out with spirit and binodid (1 to 1000) and any clots or portions of necrosed tissues are removed, no internal dressing whatever being used. The tube is next reinserted and a fresh dressing of gauze applied. The stitches are usually left until the second dressing, which takes place on the day but one after the first, when a similar procedure should be carried out. Afterwards the wound is dressed every alternate day. At the end of fourteen days the tube is omitted and a plug of wool loosely rolled and soaked in alcohol and boric acid (10 grains to the ounce of half-strength alcohol) is inserted in its place. This plug requires changing every six hours.

(b) Heath's method is practically the same as for this conservative operation (see conservative operation). The stitches are removed the day after the operation. A "horse-shoe" shaped cotton wool plug soaked in iodoform emulsion is inserted in the antrotympanic cavity lying across the opened aditus—one end in the antrum, the other in the tympanum.

The plug should be of sufficient size and molded to closely fit these cavities. A rubber tube in diminishing sizes is retained until healing is complete, and the ear dry. Dressings are changed daily.

II. METHODS WITHOUT A TUBE.

(a) Dry method. Dressing without tampons was recommended by Zarniko[20] in 1898, and later by Muhlen, Stein and others. At the completion of the operation the wound is dried with alcohol, dusted with aristol powder and a strip of gauze is placed in the meatus to act as a drain. The first dressing is done on the third day, when the wound is cleansed with drops of hydrogen peroxid instilled until effervescence ceases. The excess of peroxid is removed with dry wool and the wound swabbed out with alsolute alcohol, and again dried. Aristol powder is then insufflated, and a piece of ribbon gauze placed in the meatus as at the time of the operation. Subsequently this dressing is carried out daily, or on alternate days if the discharge is slight in amount.

(b) Wet method. The first dressing is done on the second or third day. The mastoid wound is syringed through the meatus with warm boracic lotion, dried, and a gauze drain inserted in the meatus. Daily dressings are necessary

(c) Plugging. It is claimed for the method of dressing by firm plugging of the bony cavity with plain or iodoform gauze, that secretion is reduced, that granulations are prevented from becoming excessive and that the meatal flaps are retained in a correct position. The first dressing, usually a painful one, is done on the fifth or sixth day after operation. The plugs, loosened by soaking with hydrogen peroxid or irrigations of normal saline solution, are removed. The wound, after inspection, is cleansed with mops soaked in a weak solution of binodid in spirit, dried, and repacked with iodoform ribbon gauze. Afterwards the dressings are changed daily, or on every second day, according to the condition of the wound, the amount of discharge, rapidity of repair and the comfort of the patient.

III. VARIOUS METHODS.

Skin grafting. Among the methods adopted with the object of shortening the period of epidermization of the mastoid cavity, skin grafting has taken a prominent position. It has

been recommended by Reinhard,[2] Jansen, Ballance,[3] and Politzer.[4] The methods carried out by these different surgeons vary. Ballance, after performing the ordinary radical mastoid operation, reopens the postauricular wound in ten days' time, and applies large Thiersch grafts (obtained from the forearm or thigh) to the granulating mastoid wound. Jansen inserts the grafts at the time of operation, using small ones, with which he papers the surface of the mastoid cavity, covering them with gauze smeared with boric ointment. Politzer transplants the graft, which measures 1 to 2 sq. cm., between the sixth to twentieth days after operation, the period depending on the rapidity of the formation of granulations. By means of a special canula the graft is blown into position through the meatus, the whole cavity being then plugged with small, sterilized cotton wool pledgets. Union is said to take place within three to five days.

The principal objections to skin grafting are that: (1) Two operations are necessary, i. e., the application of the graft. (2) Disease may continue in the bone beneath the flap, and being hidden is not accessible to treatment, as in a case that I have observed recently. (3) The period of healing is not materially reduced. (4) The hearing power is greatly impaired—from the covering up of the footplate of the stapes.

Blood clot dressings. Blood clot dressings were introduced by Blake[5] in 1891 as being useful in Schwartze operations undertaken in cases of acute mastoiditis. His method is as follows: The mastoid antrum and its accessory cells are opened in the usual way, care being taken to remove all the disease, paying strict attention to asepsis. The drum is then freely incised to allow of free drainage from the tympanum. The cavity left in the mastoid process is allowed to fill with blood obtained from the edges of the wound—the incision being then closed with sutures. A small opening is left at the lower angle of the incision to allow of the passage of serum from the clot. The skin incision heals by first intention. A probe is passed daily into the clot through the opening left behind the ear to aid the escape of serum. Blake claims for this dressing: (a) Blood clot with its serum is protective (i. e., even if the wound is septic), for at least forty-eight hours after operation. (b) Blood clot is a scaffolding for the formation of dense fibrous bands in the mastoid. (c)

Even should the clot break down it has aided the formation of healthy granulations.

Sprague,[6] also dealing with blood clot dressings, says 68 per cent of cases treated with this method were successful, being healed in from seven to fifteen days. He claims the period of healing is lessened, pain is reduced, and scar is less noticeable.

Serum dressing[7] is a modification of dressing by "blood clot," and was introduced by Mr. Stuart-Low. The method of its use is as follows: When the operation is completed the cavity is carefully dried and fresh horse serum (B. W. & Co.) poured into the wound. A strip of gauze soaked in serum is then passed to the bottom of the cavity. An external dressing of cyanid gauze and wool having been applied, the special cage designed by Stuart-Low to avoid pressure on the auricle and to aid drainage, is placed over the dressing and retained in position by a wide bandage. Stuart-Low claims that serum (1) encourages leucocytosis; (2) supplies opsonins and antibodies; (3) encourages repair by feeding granulations; (4) avoids desiccation. Of results obtained by this method, I can speak with some knowledge, as I have been privileged to observe many of his cases and they have been as satisfactory as any radical operation I have seen.

Vaselin dressings. Roy[8] recommends dressing the wound made in the radical mastoid operation with strips of plain gauze, soaked in sterile liquid vaselin. The first dressing is done about the sixth day after operation, the later ones are carried out daily, or on every second day. He claims for the dressing: (1) That it is less painful; (2) that granulations do not form as readily; (3) that epidermization of the cavity is more rapid; (4) that the cavity retains its original shape. I have given this method of dressing a fair trial and do not find that it comes up to its originator's expectations.

Bismuth paste. (Beck.)[19] A new method of dressing the exenterated mastoid process is that suggested by Beck of Chicago. The operation completed, the cavity is filled with a paste of the following composition: Bismuth subnitrate, 30 parts; vaselin, 60 parts; white wax (120° melting point), 5 parts; paraffin, 5 parts. The postauricular incision is closed, but a drain is provided for by the insertion of several strands of silk worm gut. The paste remains permanently within the cavity.

Soap emulsion dressings have recently been recommended for mastoid wounds. The composition of the emulsion is, potash soap, soda soap, olive oil, of each 1 dram; distilled water to one pint. The wound is lightly packed with strips of gauze soaked in the emulsion. This method is said to considerably hasten healing and to exert a beneficial effect in cleansing the cavity.

Picric acid dressings have been suggested by Bondy,[21] who has found them useful in cases where epidermization was slow. After careful cleansing of the parts he applies an ethereal solution of picric acid (1 to 10), every second day. The application is painful. Between the applications boric acid is insufflated. He claims for this dressing: (1) Secretions lessen; (2) exuberant granulations disappear; (3) epithelialization is aided.

GENERAL TREATMENT.

Healing completed, and the cavity lined by glistening epithelium, crusts, composed of desquamated epithelium mixed with wax, are apt to form, and require removing. It is wise, therefore, for patients to visit the surgeon at regular intervals to have these crusts removed. The following drops may be of service when this crust formation is excessive: Salicylic acid, 5 grains; spirit vini rect., 4 drams; glycerin, 4 drams. These drops may with advantage be followed by instillations of hydrogen peroxid.

While devoting his attention principally to the wound, the surgeon must not overlook the general condition of the patient. Previous to the operation the general health of the patient may have suffered as a consequence of the "running ear." During the aftertreatment, tonics and cod liver oil are indicated, and fresh air is always desirable. In general treatment the internal administration of calcium iodid, with the object of aiding epidermization of the mastoid cavity, should be mentioned. It has been recommended by Williamson[9] for the purpose of promoting the healing of broken skin surfaces, and maintaining a stimulating effect on epidermal growth. It is given in doses of five to eight grains three times a day. Personally I have not found it very successful as an aid to epithelialization in the fifty radical mastoids in which I gave it a trial. In the later cases I applied it locally as a lotion as well

as giving it internally; in these few cases it was an aid, and I propose to try it again when a suitable opportunity arises. It is very probable that the new cell proliferant "allantoin" may prove a valuable addition to the armamentarium of the aural surgeon as an aid to epidermization after operation. I propose trying it in my next radical mastoid operation.

Calcium lactate given for forty-eight hours before operation, with the object of raising the coagulability of the blood and lessening hemorrhage, is of undoubted service.

COMPLICATIONS WHICH MAY ARISE DURING THE AFTER-TREATMENT.

Acute labyrinthitis (serous). A complication not rare after the radical mastoid operation is acute serous labyrinthitis. It is probably caused by some injury to, or dislocation of, the footplate of the stapes while curetting in the tympanic cavity. The symptoms are acute in their onset, and usually appear within forty-eight hours of the operation. Severe headache and giddiness are complained of, so severe that the patient may be quite unable to sit up in bed. The vertigo is accompanied by spontaneous nystagmus, rotatory in type, and most marked when the patient is looking towards the sound side, passing off or less marked when looking to the side of the operation. There may be a slight initial rise of temperature and a considerable acceleration in the pulse rate. In my experience the various symptoms pass away gradually in the course of a few days, without resorting to any active treatment, although lumbar puncture very often gives great relief. Rarely, if ever, does this serous form become purulent, requiring drainage of the internal ear.

Acute adenitis. An acute inflammation of the glands of the neck is not uncommon, especially in tuberculous children, in whom it appears to be an acute exacerbation of a chronic condition. The glands most often involved are those lying under cover of the sternomastoid muscle. Occasionally they may break down and a cervical abscess form, but usually the adenitis may be kept in check by the use of lead and opium lotion, a belladonna plaster, and the internal administration of the syrup of the iodid of iron.

An enlarged gland at the stylomastoid foramen has been

known to press on the facial nerve as it emerges and cause the gradual development of facial paralysis.

Conjunctivitis is also met with in young children after these operations. Children are apt, when the wound is irritable, to put their fingers under the bandages in the attempt to scratch the wound, their fingers and nails become infected and infection may thus be carried to the eye. The resulting conjunctivitis should be treated on the usual lines. Boric lotion washes frequently, and drops of argyrol 10 per cent with the "ice pack," are of use.

Erysipelas is now rarely met as a surgical complication, although in those who are liable to attacks of this disease, it may occur during convalescence from the mastoid operation without having any relation to the wound. In one case that was under my care the erysipelas commenced on the auricle of the opposite side, and was most likely induced by the moist sodden condition of the epithelium of the part from constant lying on the one side. It is only right to say that the convalescence had been an unduly protracted one and in addition there had been some considerable fever. Erysipelas occurring during the healing process does not of necessity delay the healing. In several cases that I have seen, accompanied with high fever, the infection has had practically no effect upon the wound. In fact, it rather appeared that repair was hastened, certainly it was not retarded. The appearance of the wound, the temperature, and the other symptoms, require no description; but as to the treatment, I consider a vaccine prepared from the wound the most efficient means of combating the disease, for I have obtained excellent results by this method in seven cases. Stucky, who has made a study of erysipelas as a complication of the after-treatment of mastoid operations, believes the occurrence of this disease during the healing process to be due to autoinfection from the nose or accessory sinuses, and quotes eleven cases[29] in support of his theory. The duration of the disease appears to be self-limited to eight to twelve days and, he says, causes "no apparent delay in the healing process." As regards treatment, he recommends the local application of a paint composed of equal parts of carbolic acid, camphor and olive oil.

Nasal disease may be responsible for a certain number of cases of erysipelas, but cases occur when no disease of the nose

can be found. I have recently seen three such cases, one in a patient liable to the disease, one as a result of irritation of the skin from hot fomentations, and one which could be ascribed to nothing beyond a very acute streptococcal infection. These three cases were all successfully treated with antistreptococcic serum (polyvalent). There was no retardation of the healing process. George Leland reported a case where a mastoid operation had been performed in a case of primary erysipelas, and healing took place as promptly and as thoroughly as it would have done through the normal skin, and the erysipelas remained external.

Unguentum ichthyol smeared over the affected area has been strongly recommended by Dr. Percy Jakins, but is objected to by some patients on account of its peculiar odor.

Acute follicular tonsillitis occurring during convalescence from operation is a complication which may considerably retard the healing of the ear. There is considerable reaction within the ear, inflammatory swelling and redness with great increase in amount of discharge. The infection reaches the ear by way of the eustachian tube. Pain within the ear is also usually complained of. On the other hand, failure to close the eustachian tube after operation may lead to infection of the throat from the ear, and so cause a tonsillitis. The treatment of the ear should consist of frequent dressings; several times a day is necessary, judged by the amount of discharge; the tonsillitis by sodium salicylate and calomel internally, and gargles and paints locally.

Pyrexia may be an indication that the wound requires dressing, that a "stitch abscess" has formed, or that some part of the wound is not draining freely. A slight elevation of temperature is usual after operations of this kind, as it may be after any operation, and is usually of no importance. If an iodoform dressing has been used a moderate elevation of temperature may be accounted for by an iodoform dermatitis. A sudden high elevation of temperature (i. e., to 105° to 106° F.) without local signs is indicative of blood infection. A careful bacteriologic examination of the blood should immediately be made, and on discovery of the infecting organism or organisms, a vaccine should be rapidly prepared from it and given without delay. When there is more than one organism present it is enough to use a vaccine against the

more numerous type, the others will be killed by phagocytosis.

In many septic mastoid wounds a remittent temperature of the pyemic type is sometimes developed. This appears to be due to absorption from the pressure of the dressing, plugs or tube, whichever is being used. In these highly septic conditions an autogenous vaccine should be prepared and injected as a routine treatment, the dosage being controlled by the amount of reaction and the opsonic index.

The rise in temperature occasionally observed after the cortical operation, which is often associated with a highly septic condition of the mastoid in young children, may be very successfully treated by large enemata of normal saline solution. It is also well to bear in mind that pyrexia occurring after mastoid operations may really have no relation to the wound at all, but may be caused by some other disease, e. g., influenza.

Delayed healing may be due to innumerable causes, general, local, and constitutional. The general and local causes will be considered separately. The chief constitutional causes of delay in healing are tuberculosis, syphilis, and diabetes. The tubercular wound is characteristic, with its pale, flabby granulations, indolent edges and little sign of reaction or healing. When one is in doubt as to its character a scraping should be taken and examined, for it must be remembered that the tubercle bacillus is never found in the discharge. The treatment giving the best results consists in fresh air, cod liver oil, and the dressing of the wound with iodoform emulsion. Tuberculin injections in some cases have given satisfactory results, but, speaking generally, the action of tuberculin is somewhat uncertain.

The syphilitic wound is not so typical. It is only when one's suspicions are aroused of constitutional syphilis that an explanation of the red, indolent, nonhealing wound is found. The administration of iodid of potassium, in increasing doses, is indicated.

The diabetic wound presents no peculiar qualities beyond tardiness of repair. It is needless to say that no operation should be undertaken in a diabetic subject unless absolutely necessary in order to save the life or the hearing.

Other conditions, such as thrombosis of the lateral sinus, brain abscess and meningitis, are sometimes met with, and

complicate the after-treatment. They occur more often before operation, and the surgeon must be on the lookout for such danger signals as headaches, rigors, sickness, and when they occur their cause should be sought for and the condition, whatever it is, treated on the lines laid down for these various diseases.

LOCAL COMPLICATIONS—EXTERNAL TO THE WOUND.

Eczema of auricle and meatus is liable to occur when one has to deal with a scanty, concentrated offensive and therefore irritating discharge; this is often observed in dirty children of the poorer classes. Another cause of irritation is the use of strong antiseptics, especially in the form of medicated gauzes. The remedy is simple, and dressings of plain aseptic gauze should be used combined with some soothing ointment.

Perichondritis of the auricle is a rare complication following the radical mastoid operation, yet when it does occur it is a troublesome one and usually results in great deformity.

Politzer[10] states "that it has been frequently observed following mastoid operations of the plastic type, and is usually associated with bacillus pyocyaneus, and is not due to injury of the cartilage." The bacteria which I have found in five cases were streptococcus in three cases, staphylococcus in one case, and bacillus proteus vulgaris with the bacillus subtilis in one case. The onset of a perichondritis may be ushered in by a rigor, and with a considerable rise in temperature usually, or there may be no temperature rise at all. In several cases which I have observed it commenced with what was apparently an erythema. There are all the signs of an acute inflammation, i. e., redness, swelling, heat and pain. An examination of the blood reveals a very high degree of leucocytosis, indicating a virulent infection.

Treatment in the initial stages should consist in the application of a "cold coil" or an evaporating lead and opium lotion. Later, hot fomentations, and the frequent puncture (daily) of the auricle with a hypodermic needle are of service. Incision, for the relief of tension, is usually necessary and should not be delayed even if in doubt as to the presence of pus. An autogenous vaccine I have found to be a valuable aid in the treatment of these most undesirable cases.

Displacement of auricle in a downward and forward direction may result from faulty apposition of the edges of the

wound, or to some interference with the suspensory structures of the auricle. Undue prominence of the auricle is much more frequently observed, and is mainly due to forward pressure of large postauricular dressings. Careful replacement and bandaging of the ear is, as a rule, a sufficient remedy, but in extreme cases a plastic operation, i. e., removal of a lozenge-shaped piece of skin at the junction of the skin of the auricle with the skin of the mastoid process, including the operation scar, may be required.

Edema of face, eyelids, and temporal region is occasionally observed to follow operation, and may come on within twenty-four hours. It may be due to sepsis in the upper part of the wound or too tight a tube. If due to the former, that part of the wound should be opened for drainage; if the latter, the remedy is obvious.

LOCAL COMPLICATIONS—WITHIN THE WOUND.

Secondary hemorrhage may occur, but it is rare. Firm pressure with a large pad of wool will control it, or if preferred the wound can be plugged for twenty-four hours with ribbon gauze soaked in adrenalin 1 to 1000.

Facial paralysis may be due to other causes besides the chief cause and the most frequent one, injury at the time of the operation; (a) contusion of the nerve may give rise to a partial paralysis developing some hours after the operation; (b) compression of the nerve may be produced by the tight plugging of the cavity with gauze, though only if the nerve is already exposed, from the use of too large a tube, or from one imperfectly cut at its inner end, allowing pressure to be exerted at a part where there should be none. The onset in these instances is usually insidious, and is generally observed within twelve hours of the operation. The proper method of cutting the tube is described under treatment. If the aqueductus fallopii be partially destroyed and the seventh nerve exposed, the nerve may be compressed in scar tissue during healing and cause the slow, gradual development of facial paralysis.[12] The prognosis, as regards recovery of the paralysis is good, for practically all the cases recover if the cause of the pressure is removed at once; (c) inflammation—neuritis of the seventh nerve usually develops in from two to five days. It is rarely complete, involving only those fibers which supply the upper part of the face.

Complete idiopathic paralysis may occur apart from any mastoid operation, as in a case reported by Hahn,[11] in which paresis occurred a few hours after such an operation. The patient subsequently died from an accident, and the postmortem examination revealed a neuritis of the seventh nerve.

The treatment of all these forms consists, first, in the removal of the cause, if possible; secondly, keep up the nutrition and functional activity of the muscles and nerve by the use of the galvanic current (massage may also be of use in this respect); thirdly, the internal administration of strychnin and iron in the early stages, and iodid of potassium in the later.

Stitch abscesses occasionally occur. There is no difficulty in their diagnosis—the redness of the edge of the wound combined with some swelling and a slight rise in temperature, are sufficient to indicate the nature of the trouble. It usually clears up after one or two boric fomentations. The offending stitch should, of course, be removed. Very rarely one finds the whole postauricular wound break down—when this happens the wound should be irrigated with some mild antiseptic lotion such as boric lotion, plugged with plain aseptic ribbon gauze and kept open until there are signs of granulating. Michel's metal sutures are then of great service in bringing together the edges of the wound.

Excessive granulations and formation of polypi. After any mastoid operation a cavity is left, which is more or less a bony one. It is usual in all these operations to try and hasten the healing process by opening out a part of the cartilaginous meatus in the form of a flap, yet even with this assistance a considerable portion of bone remains uncovered. Immediately after the operation the portion of the cavity not occupied by plugs or the tube fills with blood and serum, which is usually entirely removed during the earlier dressings. Granulations may be expected to form in from seven to ten days after the operation on the portion of bone which the flap does not cover, and it is desirable that these granulations should spread evenly all over the bony surface, until finally they become covered with squamous epithelium which has grown over them from the edge of the flap or other part of the meatus, thus the cavity heals. At any time during this healing process, though generally at an early stage, these granulations may take on an excessive growth. There are two or three places where this is particularly prone to occur; these places are the remains of the facial

ridge, the roof of the aditus, the posterior superior part of the antrum, and the edge of the meatal flap, where it lies on the lower part of the enlarged meatus.* A reason for excessive growth at these areas is to be found in their greater vascularity. At each dressing the whole wound should be carefully examined while well illuminated by artificial light reflected from a forehead mirror through a specially large speculum, and any sign of exuberant granulation promptly suppressed by astringents. Granulations springing from the facial ridge and adital roof, if unchecked, are liable to coalesce, thus forming a bridge and later an obstructive connective tissue septum. In a similar manner if neglected a diaphragm may form at the entrance to the opened antrum and prevent the direct exit of discharge. Delay in drainage means irritation and consequently an augmentation in purulent products. Complete meatal obstruction is rare, though if it be allowed to occur the pent-up discharge will find exit in other directions and may even cause a fistulous opening behind the ear. It is thus important, during the operation, to remove enough of the posterior wall of the bony meatus to insure that there will still be room for the exit of discharge even if a diaphragm should form. In other cases where the antrum has become shut off by granulations, septa, or faulty position of the flap, cholesteatoma may form within and give rise to symptoms of cerebral pressure. Such a condition has come under my observation fully two years after the performance of a radical mastoid operation. This patient complained of constant headache, giddiness, and meatal discharge. On examination the meatus was found to be filled with firm granulations, and a fine probe could with difficulty be passed in the direction of the antrum. The reopening of the mastoid revealed a cavity the size of a small walnut, filled with cholesteatoma, and on its removal the dura mater was found to have been in contact with the fatty masses, which had also pressed on the membranous wall of the lateral sinus.

If the operation has been performed so that the whole cavity is within view during the aftertreatment, any tendency to exuberant granulations can be seen at once and suppressed. A general excess may be met by swabbing out the cavity with a solution of chlorid of zinc 30 grains to the ounce, copper

*The flap never reaches the true antrum.

sulphate 40 grains to the ounce, or plugging the whole cavity with small cotton wool mops soaked in absolute alcohol. Localized granulations can readily be reduced by the application of a crystal of blue stone, or a fused bead of mitigated nitrate of silver on the end of a copper probe. Silver nitrate, either in the form of lunar caustic or in a 50 per cent solution, is, next to the use of the sharp curette, the most efficient means at our disposal of dealing with exuberant granulations. Before applying any caustic it is well to plug the wound for a few minutes with a solution composed of equal parts 20 per cent cocain and adrenalin 1 to 1000. This diminishes vascularity and shows up the troublesome areas, and at the same time renders the proceeding less painful to the patient.

Abundant or offensive discharge is not necessarily an important complication, though it may be an indication of undiscovered disease, a tiny sequestrum, or a highly septic condition of the wound. As previously mentioned, an excessive discharge may result from too much "burring" at the operation. Excessive discharge becomes more serious if persisting beyond the normal period of repair after these operations, i. e., beyond two or three months. Under such conditions the wound should be closely searched for the presence of a pocket or recess which for some reason is not draining freely. If found, such a cavity should be curetted and plugged; its orifice thus kept open, it will then be assisted to granulate from the bottom. Areas of necrosed bone are best left until they are loose; do not attempt immediate removal on their discovery, unless you are prepared to do an extensive operation with a general anesthetic.

In an earlier paragraph I mentioned the necessity of always making certain that the ossicles and all fragments of bone are removed at the time of the operation. These are sometimes the cause of a persistence of the discharge, and in cases where an excessive, persistent discharge continues, they should be sought for. For continued discharge, if it be due to a superficial necrosis such as follows the use of the burr, a useful preparation is "enzymol" (Fairchild). This is a "purified solution of the proteolytic enzyme extracted from the fresh stomach gland, especially prepared for external application." It appears to be a solvent for pus, broken down tissue, necrosed bone, etc., by virtue of its proteolytic power. It certainly has a remarkable effect in these cases. The

method which I have adopted when using it, is, first to gently irrigate the wound with a solution of equal parts enzymol and warm water, and then to plug the wound with cotton wool soaked in a similar solution. These plugs should be changed daily. Occasionally I have given drops of the same strength for the patient to instill into the ear twice a day. Under such treatment I have observed the rapid cleansing and granulation of some most septic wounds. In one case in which there was a continuance of suppuration around the tympanic orifice of the eustachian tube (in an otherwise dry cavity), I was able to check it and insure recovery by daily injecting through the eustachian catheter a 50 per cent solution of enzymol.

When the discharge is merely offensive an improvement may follow the use of plugs soaked in a solution of chlorinated soda—though on account of its irritating nature it is necessary to protect the skin by vaselin. Among other drugs occasionally found useful under such conditions may be mentioned a weak solution of bichlorid of mercury, perhydrol (Merck), salicylic acid in solution,[13] formalin, 20 per cent (which has the disadvantage of retarding the granulating process).

One occasionally meets with wounds which are deficient in granulations and abnormally dry. These cases are often tedious, though Wright's hypertonic solution may be effective "for unchoking a blocked filter by setting up an outward lymph flow." This solution has the following composition: Sodium chlorid from 1.5 to 4; sodium citrate, 1; distilled water, 100. The wound is syringed with this solution, and wicks saturated with the solution are afterwards packed into the cavity, the surrounding skin having been previously smeared with vaselin. Phagocytosis* should be tested at regular intervals—if excessive it can be controlled with calcium chlorid, 1; precipitated chalk, 400; applied locally. Dressing every six hours is necessary with this treatment. The results obtained in two cases were so encouraging, that I have tried it in several of these "dry cases" with equally good results.

Stenosis of the meatus, as a complication of radical mastoid operations, is by no means rare and is more often due to inadequate care in the aftertreatment than to all other causes com-

*The phagocytic action of the wound as tested by examination of the discharge under the microscope.

bined. Though it may be due to a faulty position of the flap, to a badly cut flap or to effusion between the flap and the bone, yet it often occurs later and may be due to an excess of granulations, which, after having become organized, have contracted. In those cases in which a tube has been used, contraction may occur from its too early removal. I have never seen contraction follow when the tube has been retained long enough. Stenosis may even result from a chronic eczematous inflammation of the walls of the passage. This inflammation may extend to the deeper parts of the meatus and involve the bone, causing a chronic periostitis such as is frequently found in chronic suppurative catarrh of the middle ear, and generally spoken of as "hyperostosis of the meatus." I do not refer to the gouty forms of hyperostosis, but to those that are a sequence of a "running ear." When allowed to occur during the granulating process, discharges may become obstructed and dangerous symptoms arise, as in one case which comes to my mind, that of a patient who, after the radical operation, had neglected to attend hospital for some time, although still having a meatal discharge. When at last he came, several months after the operation, he stated that the discharge had gradually diminished. Associated with this diminution there arose symptoms of pus retention in the mastoid cavity. There was mastoid pain, frontal headache, occasional vomiting and stiffness of the lower jaw. The pain was very severe and had kept him awake for several nights before coming to hospital. On examination an almost complete stenosis of the meatus had occurred. Later, on the operating table, a large abscess was found in the antrotympanic cavity. "Prevention is better than cure," and stenosis should never occur if operative details are considered and the wound receives proper attention after the operation.

Inaccessibility of the antrum is more often due to a badly designed operation than to neglect of the aftertreatment, although only discovered then, and may not be evident even at the first dressing. A swollen flap may be the cause, or it may be due to an insufficient removal of the posterior wall of the bony meatus, or the opening into the antrum from the meatus may be too small. Such faulty arrangements may cause much trouble and even necessitate the reopening of the wound.

Keloid involving the postauricular scar is a rare though

painful complication. Fibrolysin injections have no effect upon it; the only treatment which is effective is to remove the scar and bring the edges of the wound together afresh.

Cicatricial abscess. An abscess in the cicatrix is a rare sequel occurring after mastoid operations. The scar becomes red, swollen and tender. The inflammation spreads to the surrounding parts and an abscess soon forms which may burst and leave a discharging sinus. The cause may be: (1) an abrasion of the surface and subsequent infection of the part; (2) the presence of a small subjacent sequestrum; (3) recrudescence of the old middle ear infection, rapidly extending to the surface. Usually one or two fomentations are sufficient to bring about healing, unless the abscess is due to fresh disease about the mastoid process, when a reopening of the whole wound becomes necessary.

Mastoid fistulæ occasionally occur after mastoid operations —cortical and radical. They are generally due to incomplete removal of the diseased mastoid tissues or to interference with drainage. Such a fistula may be left, in cortical cases where healing has taken place by granulation from the bottom, to an ingrowth of surface epithelium. This may often be induced to close by destroying the epithelial edges with a caustic such as trichloracetic acid, or by the galvanocautery. Some cases may be so severe as to require a plastic operation, and if the fistula be due to carious bone, a complete reopening of the wound may be the only remedy.

Implantation dermoid cyst is an extremely rare complication and occurs in the scar of the operation. A case of this description was described by Dr. Abercrombie,[14] another was shown by Dr. Hunter Tod[15] at the Otological Society.

PATHOLOGY OF POSTOPERATIVE DISCHARGES.

A. Bacteria. Organisms found in one hundred cases in order of frequency: Staphylococci 23, bacillus proteus vulgaris 15, streptococci 11, bacillus subtilis 8, cladothrix 4, spirocheta fetida 4, pneumococci 4, pseudodiphtheria bacillus 4, bacillus fetidus 4, bacillus butyricus 4, bacillus pyocyaneus 4, diplococcus catarrhalis 4, bacillus coli communis 4, acid fast bacilli 3 (not tubercle), Klebs-Loeffler 3, tubercle bacilli 2, bacillus hastilis 2, penicillium glaucum 2, mycelium 2, micrococcus tetragenus 2, torulæ 2, bacillus crass 2, Gram negative bacilli (not known).

B. Cells. Leucocytes. Poly- and mononuclear in all discharges.

Lymphocytes indicative of a healthy granulating surface. Usually found in the proportion of three lymphocytes to four leucocytes. In acute conditions this proportion is reduced to about one in twenty.

Myelocytes. The presence of these cells in a discharge is suggestive of bone disease, such as small sequestrum or tubercle.

Plasma cells. Indicative of chronic or nonleucocytic inflammation.

In regard to relationship of organisms present—character of wound, and results obtained, in the meantime I have been unable to arrive at any definite conclusions.

RESULTS OF RADICAL MASTOID OPERATIONS.

In considering the results obtained in 260 radical mastoid operations I have divided them into three main groups for purposes of analysis.

Table A. Duration of suppuration and completion of healing.

Table B. Age of the patient and results as regards cure of the suppuration.

Table C. Duration of the suppuration and results as regards state of hearing after operation.

TABLE A.

Duration of suppuration and completion of healing.

Duration of Suppuration.	Healed in one to two months	Healed in two to four months	Healed in four to six months	Healed in six months to one year	Healed in one year and over*
One month to six months..	22	26	6	0	14
Six months to one year...	8	10	1	5	0
One to five years.....	3	21	1	1	17†
Five to ten years.....	10	6	3	1	23
Ten years and over.....	7	25	7	1	42

*Over one year includes cases still discharging.

†Included in this number are two cases of thrombosis of lateral sinus, and one of stenosis of the meatus.

TABLE B.

Age of the patient and results as regards cure of the suppuration.

Age of patient at time of operation.	Ear still discharging	No discharge.
Years.	Cases.	Cases
1-5	10*	24
5-10	7	27
10-20	23	48
20-30	14	52
30-40	19	8
40-50	19	3
50-60	3	0
60 and over	3	0
Total.....	98	162

TABLE C.

Duration of the suppuration and results as regards state of hearing after operation.

Duration of suppuration at time of operation.	Hearing improved by operation.	Hearing diminished by operation.
Months.	Cases.	Cases.
1-6	0	7
6-12	10	9
Years.		
1-5	29	46
5-10	17	10
10 and over	61	30

The conclusions which can be drawn from the above figures are:

1. The sooner a chronic suppurating ear is operated upon, the better is the chance of recovery.

2. That even a very chronic ear discharge can be cured.

Forty-one cases showed no alteration in the hearing.

Thus in 117 patients hearing was improved by the radical operation, and in 102 patients it was rendered worse.

It will be seen from the above table that deaf patients with suppurating ears stand a chance of obtaining improved hear-

*Includes three cases of tubercular disease of mastoid.

ing by operation—even in the most chronic cases, although the safety of the patient's life should be the first consideration, and preservation of the hearing a secondary one.

In this connection Politzer[16] states, "that patients with considerable hearing before operation usually notice a change for the worse following the radical mastoid operation."

He mentions Stacke's figures—100 cases. Hearing diminished in 6, increased in 31, unaltered in 63.

Schwartze states that "if the labyrinth was intact the hearing is usually improved." In 71 cases operated upon, the hearing was improved 39 times, unaltered 28 times and made worse 4 times. Regarding the cure of the suppuration Schwartze quoted 200 cases, of which 99 were cured, 37 not healed but "dry," 34 unhealed, 19 unaffected by the operation and 11 died. Of the 11 deaths only one died from a postoperative complication (i. e., meningitis).

Körner also believes the hearing is improved by the operation if the labyrinth remains intact.

Grunert's figures, quoted by Hammerschlag, are the reverse of Stacke's. Grunert states after the radical mastoid operation 55 per cent of cases showed improved hearing, 6 per cent were worse, and there was no alteration in 39 per cent.

Oppenheimer[17] states that granted the labyrinth is intact, the hearing is not rendered worse as a result of the radical mastoid operation, but may be improved.

Grossmann's figures are interesting—212 cases were examined. Improvement in hearing after the radical mastoid operation noted in 93 cases (43.8 per cent). Diminished hearing in 51 cases (24.1 per cent). No alteration in 68 cases (32.1 per cent).

My own conclusions drawn from a large number of these operations are, granting the labyrinth is intact before and after operation:

1. Great deafness before the operation is usually improved by the operation.

2. Slight deafness before the operation is always made worse by the operation.

3. Duration of the suppuration previous to operation has little influence on the power of hearing after operation.

4. The quicker the healing, the better is the prospect of obtaining an improvement in hearing.

5. Hearing depends on the adhesions and density of the scar tissue about the round and oval windows.

6. Hearing is always worse if the stapes be injured or displaced at the time of the operation.

AFTER-TREATMENT OF OPERATIONS FOR TEMPOROSPHENOIDAL BRAIN ABSCESS.

The abscess having been found and evacuated through a separate opening, a large perforated rubber drainage tube is passed into the bottom of the cavity, the parts around being packed with plain ribbon gauze. If much oozing takes place the outer dressings may, with advantage, be changed the following day. The next day (second after operation) the tube may be cleared with dressing forceps, but should not be removed until the fifth day after the operation. On removal of the tube the cavity will be found to have contracted considerably, and it is in this that danger lurks. The intracranial pressure is constantly tending to press the surfaces of the cavity together, and unless great pains are taken to search for pockets or recesses these may form and become the seat of fresh trouble. The pulse and temperature must be carefully watched and studied along with the other clinical features and the appearance of the wound. For the inspection of the deeper parts of the abscess cavity a Green's dilating nasal speculum may be found useful. The drainage tube may now be shortened by one-fourth of an inch and reinserted with the plugging around. In place of the tube Neumann recommends the introduction of strips of iodoform gauze soaked in hydrogen peroxid. Daily dressings are most successful, and at each dressing the drainage tube may be shortened. "The shortening of the tube is carried on till at the end of ten days its intradural part will probably be reduced to a length of one-half inch, its caliber being always maintained" (Scott and West).[23]

The tube may now be dispensed with. Healing is usually complete in six to nine weeks. Granulations are liable to form on the dura adjoining the margins of the skin incision and may ultimately cause adhesion between these surfaces. These granulations must be kept under control and their adherence prevented. A complication not uncommon following upon operations undertaken for brain abscess is hernia cerebri. When a tendency to protrusion of the brain is noted, a firmly

bandaged dressing should be applied and endeavors made to exert a resisting pressure on the protruding part. Should this not prove sufficient and a hernia develop, firm pressure may still be maintained with the aid of a pad and a bandage. The removal of the hernia by the aid of the scapel may become necessary. The results of operations undertaken for brain abscess are very varied. According to Körner, 50 per cent of his cases operated upon, recovered. Meningitis is the most frequent cause of death. Fatal results may also be due to diffuse encephalitis, acute edema of the brain, metastatic abscesses, meningitis, pyemia and general paralysis from pressure on the medulla oblongata (Politzer).[24]

AFTER-TREATMENT OF OPERATIONS FOR CEREBELLAR ABSCESS.

The after-treatment of these operations does not vary to any marked extent from that described for abscess of the temporosphenoidal lobe. The tube is the method giving the best results, the cavity around being lightly packed with ribbon gauze. The dressings are changed on the second day and the tube on the fifth. Daily dressings are then necessary. The complications which may arise are similar to those described for temporosphenoidal abscess.

AFTER-TREATMENT OF OPERATIONS FOR THROMBOSIS OF THE LATERAL SINUS.

It is usual when the lateral sinus has been explored for septic thrombosis, and which, if found, removed, to leave the postauricular incision open for some time. Otherwise the wound is treated in the same way as an ordinary radical mastoid wound.

Should the thrombus be a septic one, there may be a remittent temperature for twenty-four hours after the evacuation of the septic clot. This need not be a cause of alarm. A healthy thrombus soon becomes absorbed, as in one of the three cases I reported some time ago, where a completely thrombosed sinus was restored in fourteen days.[25] The operation completed and the sinus cleared out, it is then plugged with iodoform ribbon gauze, care being taken, if possible, to insert the plug between the membranous and bony walls of the sinus. This plug remains in situ for three or four days. It is then removed with great care, avoiding a direct pull on the wall of

the sinus which might disturb the clot. A fresh piece of ribbon gauze should be at hand in case of hemorrhage. The wound should not be irrigated, but if it be a very septic one, as often these septic thrombosis cases are, it may be mopped out with a little hydrogen peroxid. The sinus is then replugged with iodoform ribbon gauze, a separate plug being used for the main cavity. This is very important. Dressings are then done daily. Granulations rapidly make their appearance, and in fourteen days' time it is possible to carry out the plastic operation on the meatus and close the postauricular wound.

Should the remittent temperature continue for more than twenty-four hours after the operation, large saline enemata are useful, as is also the internal administration of large doses of sulphate of quinin. Inunctions on the back of Credé's silver ointment are also recommended (Politzer).[26]

The diet should receive attention—it should be full, rich and nutritious.

If the internal jugular vein has been ligatured in the neck the wound should heal by "first intention," and the dressings need not be disturbed for at least seven days.

AFTER-TREATMENT OF THE VARIOUS LABYRINTHINE OPERATIONS.

Following the operation there is always a considerable amount of surgical shock and also signs of labyrinthine irritation, viz., nystagmus, giddiness, and vomiting. At the close of the operation on the vestibule the cavities are firmly packed with ribbon gauze, taking care to avoid any pressure on the aqueductus fallopii. Should the facial nerve be exposed, small cotton wool tampons may be substituted for the gauze plugs. The first dressing is carried out three or four days after the operation. If the flow of perilymph (cerebrospinal fluid) has been excessive, it may be necessary to carry out the dressing earlier. The cavity is from then on dressed daily at first and later every second day. Its treatment now does not differ from that of the ordinary radical mastoid operation. The proximity of the facial nerve must ever be remembered, and pressure upon it, or irritation, avoided if possible.

The granulating process within the opened vestibule is always a slow one on account of the hard, ivory like character of the bone and its poor blood supply. Granulations are liable to form and coalesce at the margins of the various openings

into the labyrinth, and unless great care is taken the minute labyrinthine cavities may become shut off from the main cavity before granulation and epithelialization is complete. A continuous flow of perilymph may also delay healing considerably, preventing the formation of healthy granulations and irritating the wound.

In conclusion, I desire to express my thanks to Dr. Percy Jakins and Mr. Charles Heath for the use I have made of their cases.

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XXXVIII.

THE WASSERMANN REACTION AND SALVARSAN IN THE DIAGNOSIS AND TREATMENT OF SYPHILIS IN EAR, NOSE AND THOAT AFFECTIONS WITH SPECIAL REFERENCE TO THE AUDITORY NERVE.*

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THE WASSERMANN REACTION.

What I shall have to say in regard to the Wassermann reaction will be particularly in reference to the employment of salvarsan, since the technic is principally a laboratory method, and the value of the Wassermann reaction as a diagnostic measure is well established and needs no further comment. As one writer recently remarked: "Today the Wassermann reaction should be made as routine a procedure in differential diagnosis as a urine analysis or an ordinary differential blood count." I wish to say, however, that extreme care should be exercised in making the Wassermann test, because I have sent the same specimens of blood to three different laboratories, and the most reliable reported a positive Wassermann reaction, while the findings in the other two were negative. The clinical diagnosis and subsequent history proved it to be a case of lues.

In regard to the various modifications of the Wassermann reaction, it is the opinion now of the majority of observers that the original Wassermann reaction is the most reliable, and since its technic has been considerably simplified, such modifications as the Noguchi, Bauer, Dungern and others have been practically abandoned.

The Wassermann reaction is usually positive in the early stages of syphilis, but not earlier than about six weeks after the development of the initial lesion. According to Sontag, in

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the secondary stage the reaction is one hundred per cent effective, and in the tertiary from seventy-five to one hundred per cent. In latent lues of an early period, that is, within four years, according to Halberstaedter, about fifty to seventy per cent will give a positive Wassermann reaction, while after that only thirty to fifty per cent. The positive Wassermann reaction is, of course, diagnostic, since other conditions in which it is found have an entirely different clinical picture, especially in ear, nose and throat affections. Some of the conditions that give a positive Wassermann reaction are recurrent fever, leprosy, measles, scarlet fever, pneumonia, thrombosis, malaria, lead poisoning, tumors and cachexia. It is, however, difficult in the cases of a mildly positive or negative Wassermann. Here all the clinical features of a lues may be present, but the serologic test negative. This is ascribed to the mercury-fast spirochetes or to the quiescent period of these organisms, and only after a longer period of rest from mercury, and after a repetition of the introduction of that drug or salvarsan, will the Wassermann reaction become strongly positive. Gennerich and Heidingsfeld independently first showed this condition and called it the "provoking test."

Again, a negative Wassermann reaction and the absence of all clinical phenomena of lues in a patient who has had the disease, but one who had been thoroughly treated, may be followed by recurrences, especially in the nervous system, within three years. After that he may be considered cured even in the presence of a Wassermann reaction, according to Fritz Lesser, Citron, and others, while Halberstaedter and others will hear nothing of a time limit as to when a patient is cured. Noguchi recently introduced a new reaction for syphilis, which is analogous to the cutaneous reaction for tuberculosis, and is known as the luetin reaction. This is especially adapted to the latent stages of syphilis, because in many instances the Wassermann reaction is negative, while the luetin is positive. Again, it is absolutely negative in a healthy as well as cured syphilitic. So that, according to Noguchi, when the allergy (skin reaction) does not occur and the patient has also a negative Wassermann after having been treated, he may be considered cured, regardless of time.

A Wassermann reaction may remain positive in spite of antiluetic treatment for a very long time, and this is explained

on the basis that neither mercury nor salvarsan has reached all the spirochetes. The latter are located in structures that are penetrated with difficulty by these remedies; as, for instance, the cerebrospinal system, nerves and cornea. Especially is this true when small doses are given, whereas one large dose of salvarsan transforms the positive Wassermann reaction into a negative one. It has been demonstrated that the later the antiluetic treatment is begun in a case, the longer in proportion will it require to change a positive to a negative Wassermann.

In the examinations of the cerebrospinal fluid it has been shown in quite a number of instances by the Appelt-Nonne test that this fluid has been infected with lues, while the Wassermann reaction of the blood was negative, and correspondingly in cases of faintly positive Wassermann reaction this test of the cerebrospinal fluid may be strongly positive.

SALVARSAN.

The subject of the application of salvarsan in general has been so thoroughly exploited and references in the literature are so numerous that I shall not go into this part with any great detail, but shall confine my remarks principally to otolaryngologic matter, with special reference to the complications or recurrences, and especially those of the auditory nerve.

In the January number of *Chemotherapy*, the references and reports which have been compiled from the principal nations of the world, show that from July, 1911, to October, 1911, four hundred and ninety-five articles have appeared, among which America leads with 156, France 138, Russia 73, Germany (and this is noteworthy) 66, England 31, and Italy 31. Since that time the reports have materially increased. So far as the literature on salvarsan in otolaryngology is concerned, reports of complications in the auditory nerves preceded those of the great benefits from this remedy. Chiari and Gerber were the first to report cases, and the interesting points they brought out were that threatening laryngeal obstruction, where one would be in fear of the action of potassium iodid, on account of a possible edema, cleared up with remarkable rapidity, when salvarsan was administered.

Felix, of Bucharest, made one of the earliest and most encouraging reports of salvarsan in otolaryngology from his

large experience. After that quite a number of isolated reports in discussions of papers on subjects other than syphilis or salvarsan appeared in this country and abroad. Dr. Thomas J. Harris, in such a discussion, was one of the earliest to report in New York on the great value of this remedy, but a definite and complete reference has not as yet appeared, although I know papers have been presented at the meetings just held of the New York State Medical Society and American Laryngological Society.

COMPOSITION OF SALVARSAN, OTHERWISE KNOWN AS ARSENOBENZOL.

$$\text{Formula:} \begin{cases} C_6\,H_3 \begin{cases} N\,H_2\ (CLH) \\ -\,O\,H \\ A\,S \end{cases} \\ \\ C_6\,H_3 \begin{cases} A\,S \\ -\,O\,H \\ N\,H_2\ (CLH) \end{cases} \end{cases}$$

According to Leonore Michaelis, it is derived from atoxyl, which contained five values of arsenic, whereas salvarsan only contains three values. On account of its phenol acid property, as well as the property of its amin, it is considered a base. It combines, therefore, with sodium hydrate, a sodium salt, and with hydrochloric acid, a salt of carbohydrates. It is soluble in water, and if exposed to air in dry form becomes oxidized and brown in color. This change adds very materially to its poisonous effect, and it is, therefore, necessary not to employ any preparation for solution except from hermetically sealed bottles or tubes. There is 34 per cent of arsenic in salvarsan.

ACTION OF SALVARSAN ON THE SPIROCHETA PALLIDA.

Schereschewsky, Hoffmann and Noguchi made it possible to experiment with this drug in pure culture of spirochetes transferred into rabbits, and it was found by Hata that their destruction followed almost immediately the injection of salvarsan. Ascoli, Pasini, Siedknid and Truffi showed that marked degenerative changes in the spirochetes followed the injection of salvarsan, and they could not be found after two to three days. Not only superficial lesions showed the disap-

pearance of the spirochete so rapidly, but Jaffe and Henck excised chancres and, after staining by Levaditti method, demonstrated their destruction in one to three days. In congenital lues Herxheimer showed that three days after the injection the spirochetes were absent from the whole body, except the lungs, where they were found in a markedly degenerated condition. Similar findings are reported by Reinke in another postmortem examination, only here it was four days following the injection. It is noteworthy that Ehrlich and Plaut have shown that salvarsan has no effect on the spirochetes outside of the body.

METHOD OF APPLICATION.

Three methods of application are known, the Weichselbaum or subcutaneous method being the first of which use was made; second, the intramuscular, and, third, the intravenous. The subcutaneous method has been practically abolished, on account of its painfulness and tendency to abscess formation. As to the choice and indications of the intramuscular and intravenous, it is still an undecided question; in fact, both methods have their advantages and disadvantages, as well as distinct indications and contraindications. I shall not go into the detail of the controversies, but will state the methods that I have or rather am employing at the present time.

Intramuscular Method.—Schindler and Neisser recommend the preparation known as Joha, which is a mixture of salvarsan in iodipin, in ampules, already prepared, and in doses of 0.4 to 0.6 of salvarsan in 1.0 to 1.5 of iodipin oil. One may prepare his own mixture by properly and thoroughly triturating the agents in their proportions and under the strictest aseptic conditions. Enough may be made for stock for a number of injections.

The technic is the same as for any other deep muscular injection. Recently I have incorporated, at the suggestion of Ormsby, a small quantity of soft paraffin, which reduces the irritation to the minimum.

Intravenous Method.—There are awo conditions that are of importance in this procedure: 1. Experience in the introduction of the needle and the general management of technic. 2. Absolute sterilization of everything that is employed, especially the water that is used as the menstruum. Distilled water is best.

It is not necessary to lay the vein bare, as Notthaft has declared; this not only prolongs the procedure, but adds to the possibilities of complications as well. It is necessary to do this only in the rarest instances and in very stout persons with small veins.

Steps of the Procedure.—(a) Preparation of the solution. About 40 cc. of warm, freshly prepared normal salt solution is poured into a graduate breaker (with a glass stopper and about twenty glass beads) measuring about 300 cc. To this is added the entire quantity of the ampule of salvarsan (0.6), and this is shaken until all the yellow crystalline substance is dissolved. Add further about 200 cc. of the salt solution and shake again. Then, with a dropper, add about 20 minims of a 15 per cent sterile and filtered sodium hydrate solution, which produces a cloudy solution. The sodium hydrate solution is added drop by drop until the whole mixture becomes clear. The remainder of the 300 cc. of the salt solution is now added and the whole solution well mixed; if the least cloudiness is present, some more of the sodium hydrate is to be added. A litmus paper test will show it to be either neutral or slightly alkaline.

All three different solutions have been used—acid, alkaline, and neutral, but it is the latter that is most frequently employed. Duhot claimed a quicker and more permanent action from acid solution, but this has not been substantiated by others.

(b) Apparatus.—There are more than forty types, many very similar, that I have been able to find suggested, but the main principles are those of any transfusion syringe. The container should have a capacity of about 300 cc., held on an iron stand that may be varied in height so as to regulate the pressure. There should be a firmly secured rubber tubing not too long and wide, at the end of which is attached the canula needle, which has a three point stopcock. To one of the other nipples of this stopcock is attached another tube, going to a second container, which also rests on the stand and is for the purpose of flushing the vein with normal salt solution before and after the injection of salvarsan. The third opening of the stopcock is to determine that one is in the vein by the escape of a little blood, which is eventually taken at this time for a Wassermann test.

(c) Injection.—Both the containers are to be filled, one with

salvarsan, and the other with salt solution. Patient is put in the recumbent position. A rubber tube is applied around the uppermost part of the arm, but not too firmly, because slight paresis of the musculospiral nerve has been observed and considered a complication of salvarsan administration. One of the prominent veins is to be selected, preferably on the forearm, because it is less inconvenient afterwards than within the elbow. The region of this vein is tapped slightly, making it more prominent (Weichselbaum). The needle is passed through the skin into the vein in a very oblique, almost flat, manner for about a half inch. The stopcock is opened so that the return of the blood may be observed. The vein is relieved by removing the Esmarch rubber tubing from the arm and about 10 cc. salt solution allowed to enter. This is followed with the salvarsan. It should take about half an hour for the entire quantity (250 to 300 cc.) to enter the circulation. At the end of this time a flow from the salt solution of about 10 cc. is permitted, the needle is removed and a light pressure bandage applied. Patient is returned to bed, not walking, and told to remain quiet for about a day.

Dosage.—At the beginning Ehrlich, when he spoke of the sterilizatio magna advised that one large dose be given, but this has not been found as satisfactory as the sterilizatio fractionata, and smaller doses only, but often repeated, are now the rule. Kromayer recommended what he called a chronic salvarsan treatment—an injection of 0.2 three times weekly until a dose of 2.0 to 3.0 was reached within four to six weeks. This method did not find great favor. The most satisfactory dosage appears to be in active cases 0.5 every third day, so that after twelve to sixteen days the treatment is completed. In the more latent cases one injection of 0.5, followed several weeks later by another, with an interval of mercurial treatment, is the rule. However, in arteriosclerotic individuals, or those afflicted with heart, kidney or nervous diseases, a very small dose, say 0.1, is given, to find out how it reacts, and then it is gradually increased. In infants the smallest dose and the smallest amounts are advised, say 5 cc., containing 0.01, and this is to be repeated four or five times. Jesioneks found that these small doses often provoked the symptoms and appeared to make conditions worse, so that Ehrlich and Weichselbaum recommended the maximum dose of 0.1 at one time. Welde showed that

even larger doses were well tolerated, and Baisch gave in a newborn, and with good results, 0.15 twice within ten days. Children from two to ten years of age tolerate doses as high as 0.1 to 0.3.

The Excretion of the Salvarsan.—According to Fischer and Hoppe, Fränkel and Grouven, Ullmann and several others, who have studied this question, there seems to be a great variance of opinion, and this is due to the difficulty of obtaining and weighing the arsenic from the excretions. Lockmann says that salvarsan, of all the other arsenic preparations, is the slowest to be completely excreted. In the first twenty-four hours after injection 0.2 to 2½ per cent is excreted. After seven and a half weeks 0.003 per cent was found in the daily urine in cases of intravenous injections, and 0.03 per cent in subcutaneous injections, ten weeks later, and 0.01 per cent thirteen and a half weeks later could still be found. Formerly it was thought that in subcutaneous and intramuscular injections the excretion of the salvarsan was much slower than in the intravenous method, but Bornstein and Lockmann have shown that this is not true, and that the liver, spleen, kidney, bone marrow and muscles serve as a storehouse for the arsenic in this as well as in all the methods.

According to Fränkel, Navassart and others, the intestinal tract as well as the kidneys are the principal structures of excretion of the arsenic, mostly from the intestines. Abelin could find arsenic as early as five to ten minutes after an intravenous injection, and in the intramuscular after half an hour. Resorcin is employed as a test for the arsenic. However, one finds that the salvarsan has not been split up or changed very much by its passage through the blood, unless, as Bornstein believes, the arsenic forms again with the urine and feces a compound much like the original salvarsan. Loeb has shown that long after the urine shows no trace of salvarsan, it may still be found in goodly quantities in the blood, and would assign this fact to the possibility of a chronic arsenic intoxication. These findings were not accepted. No one has as yet determined the lethal dose of salvarsan, but there is no question that such must exist, in spite of the fact that even after very large doses thus far given there have been no disastrous results.

INDICATIONS.

(1) Malignant or grave lues.

(2) Lues in which there exists an idiosyncrasy against mercury.

(3) Lues that is very refractory to mercury.

(4) In cases where, for prophylactic reasons, a rapid sterilization is necessary.

(5) Recurrences or socalled complications in the nervous system.

(6) In luetic patients who are also infected with tuberculosis, and who, as a rule, tolerate mercury very poorly.

(7) In cases of lues where, in spite of a very vigorous and long continued mercury treatment, the Wassermann reaction remains positive.

(8) As a diagnostic measure it is far superior in time to mercury.

CONTRAINDICATIONS.

(1) In ocular diseases in which there has been employed some of the other arsenical preparations, as hektin, enesol, arsacetin, or atoxyl, one must not employ salvarsan. In no ocular disease, especially when of luetic origin, is there any contraindication for its use.

(2) In heart diseases and aortic aneurism great care should be exercised, and only the smallest doses should be administered, if at all. In cases of heart failure complicated by nephritis, tabes, general paresis, it is, according to Ehrlich, contraindicated.

(3) Kidneys: Acute nephritis, except the luetic type, is an absolute contraindication to the use of salvarsan.

(4) Gastrointestinal: Ulcerative conditions of stomach and duodenum, owing to the possibility of rupture.

(5) Liver: Acute swelling or chronic cirrhotic or atrophic livers prove very dangerous conditions, since here is the main depot for the salvarsan.

(6) General nervous system: In all cases of marked progressive degenerative changes, especially where arteriosclerosis is present. In cases where a typical Herxheimer reaction takes place, and where the danger zone of the centers becomes affected by this reaction.

(7) Diabetes and general marked obesity, in consequence of general alcoholism, are very dangerous conditions in which to employ salvarsan, on account of the fatty heart.

COMPLICATIONS.

Complications following the use of salvarsan are not many at the present time, since the technic is very much better understood, and those conditions which formerly were looked upon as complications are in the main known as luetic recurrences, and react most beautifully to more salvarsan, mercury and potassium iodid. Especially are these recurrences or reignited syphilis manifest in the general nervous system, and principally in the peripheral nerves. The kidneys, liver, and other parenchymatous structures are next in frequency in what the Germans call "provokatorische recediven." There are, however, certain symptoms rather than complications that follow the use of salvarsan, but these are, as a rule, so transitory and leave no changes behind, that I will only enumerate some of them, and go more into detail in discussing the affection of the auditory nerve.

A group of symptoms, such as sudden redness and swelling of the face, marked difficulty in respiration, and an irritative short cough, great pressure and hot feeling in the head, the expression of great anxiety, paresthesias of the extremities, have been observed by a number following immediately the injection of salvarsan, especially intravenously. Recently, Iwaschenzow has published his experience with this particular subject and believes that we are dealing with a sort of anaphylactic condition; that the principal cause is the too early repetition and of too small doses of salvarsan, a phenomenon similar to injection of other sera. All these manifestations disappear from one-half to five minutes later. There is no change in the pulse. Epileptiform seizures, convulsions, syncope, and complete loss of consciousness have been reported. A marked rise of temperature, diarrhea, nausea and vomiting occur, but less frequently.

Some of the manifestations of the skin that were observed following the injection of salvarsan were an eruption resembling scarlet fever and measles, urticaria, herpes zoster, herpes facialis and labialis, dermatitis exfoliata, but, most interesting of all, the Herxheimer reaction. The reaction is a swelling of the tissues usually brought about by the introduction of some chemical substance (potassium iodid or salvarsan) into a body that has been previously fairly well saturated with mercury. According to this explanation, accepted by Ehrlich

and many others, the many recurrences of lues, especially in the nervous system, which will be described later, are due to this Herxheimer reaction.

As to the kidneys, hemorrhagic nephritis or simple hematuria, transitory albuminuria and slight nephritis, with granular casts, are recorded.

Marked jaundice is reported from probable affections of the liver or its gall ducts.

NEURORECURRENCE, OTHERWISE SPOKEN OF AS INJURY TO THE AUDITORY NERVE.

As otologists, we are particularly interested in three points: First, in cases of luetic affection of the acoustic nerve, is salvarsan indicated or not? Second, in luetic patients with a history of a preexisting labyrinthine nonluetic affection, are we justified in advising against the injection of salvarsan? and, lastly, and most important, is the question of the affection of the auditory nerve, either the acoustic, vestibular or both, the result of salvarsan injections?

In answer to the first and second questions the great majority of authorities agree that salvarsan should be employed, and some of the most brilliant results are reported from salvarsan in acute luetic labyrinthitis.

As to the question of auditory nerve affection following salvarsan, I will say that the authenticated literature and my personal investigations and observations, as I will show later, do not bear out those alarms that started in Finger's clinic in Vienna, and have been continually reported and rereported. In consequence thereof in many cases in which salvarsan was indicated it was withheld, and thus a better result prevented. We are thankful for these early observations of the Vienna school, and hope that subsequent observations of these cases will show that the majority of the cases proved to be only temporary, most probably due to changes which I now will describe.

There are two types of changes in the nerve possible after salvarsan injections: the acute, taking place one to two days after, and the other the latent, occurring several days or weeks after injection. Ehrlich says that the nerves are the most difficult of all the tissues to be reached by salvarsan. On the other hand, the nerves are very frequently infected by the spirochete.

Ullmann, in animal experiments with destructive doses of salvarsan, could find arsenic everywhere except the nervous system, and supports Ehrlich's contention.

The acute affections of the auditory nerve following salvarsan injection are spoken of as neuroreactions by Tröumer, and are explained as follows: The spirochetes, through a long period of mercurial treatment, become nonactive and mercury-fast, so that additional mercury will have no effect on them, and when the salvarsan is injected, it brings them back into action. Since the salvarsan cannot reach the spirochetes very well within the nerves, as has been shown above, through their action at these points certain endotoxins are liberated and there results an acute swelling of the nerve, and thus the sudden affections.

A mercurial and potassium iodid treatment, with a possible second dose of salvarsan, will clear up, as it has already done in many instances, most of these acute cases.

As to the latent nerve affection that follows two to eight months after the injection, there cannot, according to Weichselmann, Ehrlich, Benario, Guttmann, and others, be any question that these are true luetic recurrences, due to an incomplete sterilization of the whole system by one or two injections and in many instances to the inadequacy of the dose employed. In opposition to this opinion are Finger, Rille and others, who ascribe this condition as being due to the toxic action of salvarsan. Benario, in order definitely to determine the pathogenesis of this late manifestation of the nerves following the injection of salvarsan, collected 220 cases of nerve affection from the literature, and for comparison 123 cases of the same affections following the use of mercury, for the same cry was once raised against mercury as now is against salvarsan. The results of his tabulated studies are as follows:

That these nerve affections (the auditory included) are syphilitic recurrences is proven by the following:

1. They occur exclusively during the early period of the secondary stage of the disease, at a time when the dispersion of the spirochetæ is at its height.

2. They were not seen in cases other than syphilitic treated with salvarsan (exception of Heidingsfeld's case).

3. They are influenced and curable by repetition of salvarsan injections.

4. The pathologico-anatomic process, as seen in the eye fundus, is an acute inflammation, while the arsenic produces atrophic, noninflammatory changes.

That these nerve affections are not due to the toxicity of salvarsan is shown further by:

1. The long interval between injection and the first symptoms.

2. They have not occurred if salvarsan was administered correctly.

3. They have occurred where the dose of salvarsan injected was small.

4. They do not occur in cases that already show symptoms of nerve affection.

5. The same changes have occurred when mercury was employed instead of salvarsan.

The same author has found that of all the luetic recurrences in the nerves, the auditory predominates to the percentage of 44.5 per cent.

In the *Internationales Centralblatt für Ohrenheilkunde,* Valentine recently reviewed this whole subject of the auditory nerve affections following salvarsan, and comes to this conclusion: "It certainly shows that from the enormous number of cases that have been injected, but a very small number have been damaged in the region of the auditory nerve, and surely one may reconcile this difficulty with the most remarkable successes with this remedy—salvarsan." He has classified the cases into two groups: (1) Acoustic affection alone. (2) Acoustic affection with other nerves, and the acoustic nerve affection into (a) cochlearis; (b) vestibularis; (c) cochlearis and vestibularis.

There are 45 cases in all that he could find in the literature. Of these there were 9 of cochlear, 7 vestibular, 20 combined of both portions, and in 9 other cranial nerves were involved also.

Benario, in the *Muenchener medicinische Wochenschrift,* gives his collected cases from literature, and personal inquiry, numbering 14,000 cases treated, and 126 secondary nerve affections, of which 62 were of the acoustic nerve. These were divided into 29 cochlear, 5 vestibular, 17 both cochlear and vestibular, and 11 in combination with other cranial nerves.

Frey, Duel, and more recently Urbantschitsch, have expressed themselves of the belief that no permanent injury of the auditory nerve occurs following the use of salvarsan.

An interesting case of nonluetic origin is reported by Milian, in which two salvarsan injections were given, causing trouble with the auditory nerve. Since the case, however, had been previously given hektin (arsenic) for some time, it cannot be accepted as a case of arsenic injury from salvarsan. Another case of lichen planus, reported by Wanner, in which salvarsan caused some trouble with the auditory nerve, would be accepted as a case of toxic effect from salvarsan on the auditory nerve, but the report is very incomplete and cannot be accepted as such at this time. One case of O. Beck must be mentioned, in which, following salvarsan, bilateral deafness ensued. Mercury was given, without result. Then pilocarpin was given, with good result. He believes pilocarpin enhanced the excretion of the salvarsan from the system.

An interesting contribution to the subject since Valentine's review is by Knick and Zaloziec, in the *Berliner klinische Wochenschrift*: "Acute Acousticus Involvement in the Early Period of Syphilis, with Special Reference to Salvarsan." They describe in tabulated and complete form ten cases, in which they found that most of the cases where large doses of salvarsan could be given cleared up quickly; however, those of combined brain syphilis with labyrinth involvement were slow in recovering, owing to the small doses that were given by necessity. The spinal punctures showed cellular elements and in a number showed a positive test for syphilis (Appelt-Nonne), whereas in the same case a negative Wassermann existed.

In making an extensive investigation of the effect of salvarsan in treatment of syphilis in general, I have communicated either by letter or in person with forty-two physicians, most of them specialists in genitourinary and skin and veneral diseases, outside of Chicago, and herewith state my results. I wrote to physicians in the following cities: Philadelphia, Pittsburg, New York, Buffalo, Baltimore, Cincinnati, St. Louis, Boston, Washington, Detroit, Montreal, Toronto, St. Paul, Minneapolis, Toledo, Kansas City, Denver, San Francisco, Los Angeles, New Orleans, Louisville, Duluth, Jacksonville, Birmingham, Nashville, Vicksburg, Knoxville, Cleveland, Columbus, Albany, Harrisburg, Salt Lake City, Asheville, Indianapolis, Omaha, Dallas, San Antonio, El Paso.

As to complications, most of the men report very few, espe-

cially in reference to the auditory nerve. A goodly number were still very sanguine about the use of salvarsan, although thus far, when they employed it, the results were very satisfactory. The publishing of all the correspondence would probably be of interest and profit, but space will not permit it. I will, however, present the two extreme reports, and if Heidingsfeld is correct in his interpretation of his case, then it is the second case that I have been able to find in the literature of permanent injury to the auditory nerve.

"Dear Doctor Beck: "May 3, 1912.

"In reply to your letter of April 30th, I beg to state that I have just read a paper before the New York State Medical Society, at Albany, on the effects of salvarsan on the eye and ear. This will later be published in the Journal of the State Association.

"I have given the drug now about one thousand times intravenously and have seen no case where it has produced any injurious effect on the ear or eye. It is extremely important, however, that details of technic be carefully observed, the most important of which is the preparation of the drug with freshly distilled water. Sincerely yours,

"(Signed) J. A. FORDYCE,
"New York."

"My Dear Dr. Beck:

"In reply to your kind letter of April 30th, I beg to refer you to my most recent contribution on the subject of salvarsan, namely, an article which has just appeared in the May 4, 1912, issue of the *New York Medical Journal.*

"In answer to your query, I beg to state that up to the present time I have observed five cases of impaired hearing with intravenous administration of salvarsan. There was impaired hearing in two of the cases prior to the administration of salvarsan, and in two cases the impairment manifested itself subsequent to the administration. Two of these cases have proceeded to satisfactory recovery after an interval of three to four months. The other two cases have not as yet shown any material improvement. The fifth case is one of unusual interest, inasmuch as it demonstrates that the impairment of hearing must have been due to the intravenous administration of salvarsan and to no other attributable cause. This patient

had a well-defined case of scabies and presented no history or clinical evidence of existing or preexisting syphilis. The patient fell into the hands of an unqualified, incompetent physician, who admintstered salvarsan intravenously. Within two weeks after the administration the patient came to my personal notice with impaired hearing. A Wassermann examination at that time was absolutely negative in character and remained negative for a period of four weeks. The patient presented no clinical evidence of existing or preexisting syphilis. To my mind, the impaired hearing in this case must be justly attributed to the intravenous administration of salvarsan, in so far as syphilis was not associated, and it manifested itself within a few days after the intravenous administration of the remedy.

"Very sincerely,

"(Signed) M. L. HEIDINGSFELD,

"Cincinnati."

On inquiry of the Chicago profession at large, and especially the genitourinary and skin and venereal specialists, I was able to obtain the following information: From 182 men, who had used this remedy for syphilis, in very many injections and cases, there had never been a death, nor any difficulty with the auditory nerve, with the exception of slight irritation, as dizziness or ringing in the ear, always passing away within a short time. Some reported late manifestations of the auditory nerve which responded to more mercury and salvarsan, in that they cleared up.

DEATHS REPORTED IN LITERATURE.

Ehrlich and his followers claim that in not a single instance has there been a case demonstrated but what would show that the patient had, previous to the injection, changes of such nature as could explain the cause of death, independent of the salvarsan.

1. Gaucher (France). Two deaths; symptoms of meningitis; no postmortem.

2. Tucker (America). Man, thirty years. One hour after injection of 0.3 salvarsan, intravenously, symptoms of chills, vomiting, high temperature. No postmortem.

3. Goldenberg and Kolaski (America). Alcoholic. Two weeks after injection, intravenously, of salvarsan had symptoms of mania.

4. Pederson (America). Man, thirty. Brain gummi and hemiplegia, and growth in rectum. 0.45 salvarsan, intramuscularly. Twelve days later died from intracranial hemorrhage. Albumin in urine.

5. Wolbarst (America). Man, thirty-eight. Tabes. Died twelve days after injection. No arsenic could be found the first three days after injection; after that, semicoma. Before he was injected he had a mild nephritis, with albumin and granular casts.

6. Fordyce (America). Woman, twenty-five. Dead on second day after the second injection of 0.4, intravenously. A trace of albumin showed, and some red blood cells, after the first injection. A few days later, when the above second dose was given, the temperature rose very high. Vomited. This continued until the following day. Next day anuria and drop in blood pressure to 60. Autopsy showed acute nephritis and hemorrhage in the liver.

7. Longe (Italy). Boy, four years old. Symptoms of acute arsenical poisoning thirty-six hours after injection, intravenously, of 0.08 of salvarsan. No postmortem.

The cases that I have treated by means of salvarsan are as follows:

External nasal deformity combined with ulceration externally as well as intranasally	11
Intranasal, mostly ulcerative	8
Nasopharyngeal ulceration	5
Palate and mouth and tongue	6
Pharynx, including tonsils	4
Larynx	7
Larynx, trachea and bronchi	1
Ear, chronic eczema of pinna and external auditory canal	1
Ear, chronic suppuration of middle ear	2
Ear, acute labyrinthian deafness	2
	47

In all of these cases the Wassermann test showed in most instances strongly positive. All but one case had either previously or subsequent to the injection of salvarsan, mercury and potassium iodid treatment, one case for eight years.

Twenty-eight of these patients are now cured, so far as the repeated Wassermann being negative shows, and clinically all of the forty-seven cases are well. The majority were treated by the intravenous method. The majority had more than one injection, and one case, a pharyngeal lues with pathologic conditions in the eyes, fingernails and tibia of an active type, had five salvarsan (0.6) infusions within seven months, with marked benefit, but at the present time has still a marked positive Wassermann reaction. Most of the patients gained greatly in weight and improved in their general health. In three cases there was a typical Herxheimer reaction following the injections. One case, a colored woman, had all the symptoms described as anaphylactic following salvarsan, and she is the one case that had no other antiluetic treatment before or after the injection of salvarsan, and repeated Wassermann reactions since proved to be negative. This patient gained thirty pounds after the treatment within a month. Her Wassermann reaction was strongly positive before the salvarsan injection.

In not a single instance was there any trouble with the ears following the injections, except a ringing, which disappeared within a day or two.

In conclusion, I desire to state that the new preparation soon expected will be hailed with hopes that some of the objections to salvarsan will be met, and this great remedial agent will become the universally applied specific for the cure of lues.

I also wish to state that I freely used the publication of Richard Sieskind in the January number of the *Berliner Klinik*.

Since this article was read the author has treated about twice as many cases of this kind as are reported and has observed no untoward effects on the acoustic nerve from the use of salvarsan. Furthermore, no reports of ill effects from the use of salvarsan have come to the author's notice, either by personal communication or through correspondence from any of the many men who employ this method of treatment.

Referring to the case of Dr. Heidingsfeld, reported in this article, the author herewith gives a copy of a letter received from Dr. Heidingsfeld shortly after the reading of this article, relative to the case:

"I beg to inform you that the case of impaired hearing in a patient who had scabies and a negative Wassermann, has lately developed some active manifestations of syphilis, in which spirochete pallida have been demonstrated. The impaired hearing, therefore, in this case, cannot be attributed solely to the influence of salvarsan, as I first surmised."

The author wishes to make the following statement with regard to his employment of neosalvarsan, the latest substitute for salvarsan itself. It has been employed in a number of cases, particularly intramuscularly, in repeated small doses, with absolutely no bad effects, either on the nerve, nor other untoward effects attributable to the use of salvarsan. One particular advantage is its rapid solubility, but the pain caused by the injection seems to be equally as great as when using salvarsan.

XXXIX.

WHAT IS THE BEST TYPE OF RADICAL FRONTAL SINUS OPERATION, VIEWED FROM SIMPLICITY OF TECHNIC, TIME OF HEALING, AND COSMETIC RESULTS?*

By Geo. E. Davis, M. D.,

New York.

The operative measures developed in recent years, dealing with both acute and chronic frontal sinus troubles, vary greatly. Though the objective point is the same, i. e., the drainage and ventilation, or obliteration of the sinus, depending on the duration and extent of the trouble, yet each individual surgeon seems to have devised a new technic or modified an old one; all of which goes to show that, though we may have attained to a successful standard, as yet we have not achieved the ideal. This is especially so in reference to the radical frontal sinus operation.

Undoubtedly the classic Killian operation, judged by all the stipulations of the text of this discussion, barring the first, perhaps, is the best type of radical frontal sinus operation to date. But it is not perfect, and I beg your indulgence while I briefly detail yet another modification which perusal of the literature and my personal experience prompted me to make. It is meant not to supplant, but merely to supplement by simplifying the technic, and in a manner enhancing the cosmetic effect.

Citations from my case reports and the report, with photographs, of one case in particular, where I was fortunate in being able to compare the results of this modification and the original Killian in a simultaneous double radical frontal sinus operation, I trust will furnish evidence sufficient to justify the claims of my procedure.

Indications. When we bear in mind the purpose of the

*Candidate's thesis for membership in the American Laryngological, Rhinological and Otological Society.

radical operation, i. e., drainage and ventilation, or obliteration of the sinus, we appreciate that the rationale of all operative measures devised to meet these indications must be based upon a proper knowledge of the anatomy of the sinus and its relations to contiguous vital and special sense organs.

Anatomy. I shall not attempt to detail the anatomy of the frontal sinus, with which you are so familiar, further than to emphasize the importance of a thorough knowledge of its location and relations to the nasal fossæ, the other accessory nasal sinuses, the orbit and the brain; thereby to aid the interpretation of clinical phenomena in frontal sinus troubles and explain the technic of operative measures undertaken for their relief and cure.

The frontal sinuses are air cavities situated between the two tables of the vertical portion of the frontal bone, and when normal or of average size, lie immediately above the root of the nose, but when large, may extend laterally over the orbit to its external angle or posteriorly to the optic foramen. A septum usually separates them.

The development of the frontal sinuses is accomplished by an upward expansion of the anterior ethmoidal labyrinth. This process of expansion begins very early in life, probably at the end of the first year, but does not attain to a distinct, recognizable cavity, at the level of the frontonasomaxillary suture, before the sixth or seventh year, and not to full development before the eleventh or twelfth year.

Whatever may be the function of the various sinuses, which is problematic, we note in the case of the frontal sinuses, at least, that nature by developing them from below upward has provided an avenue of communication between the sinuses and the nasal fossæ, which begins at the most dependent portion of the floor of the sinus—the anterior inferior angle—and which terminates by an ostium at the upper end of the infundibulum in the majority of cases, or, slightly more anteriorly and medially, directly into the middle meatus, the channel of communication being termed the nasofrontal duct, which passes downward, backward and inward, surrounded by the anterior ethmoidal cells. These cells are contained for the most part in the anterior end of the middle turbinate and the uncinate process. The cells of the bulla ethmoidalis bound the duct posteriorly, and frequently there are one or two small

cells external to the duct, between it and the lacrimal bone. Therefore, the anatomic location of the nasofrontal duct at the bottom of the sinus is clinically important in that it furnishes positive evidence of its function—drainage and ventilation of the sinus. Furthermore, its relation to the sinus and the anterior ethmoidal labyrinth is of interest from a therapeutic and surgical view, as it is the natural, direct and simple route to reestablish drainage and ventilation, practically in all acute and subacute and many chronic pathologic conditions. But if the disease is of long standing and the mucous lining and bony walls are greatly involved, obliteration of the sinus is demanded, and it will be necessary to supplement measures, from below and within, with operative procedures directed from without. Particularly is this so where the sinus is large, when it is important, if not imperative, to have direct inspection to remove thoroughly and safely all diseased tissues.

With this brief review of the anatomic location and relations of the frontal sinuses and their ostia as a guide in interpreting clinical phenomena and to nature's way of relief, we have valuable suggestions in formulating treatment, therapeutic and surgical. To the latter we will confine our discussion.

In deciding the type and extent of operative procedures to be adapted in frontal sinus operations, not only the surgical conditions, but the patient must be considered. With private patients, ladies especially, the question of deformity is of prime importance, while with hospital cases and laborers, who may have dependent families, the time of healing must be regarded and the question of deformity relegated to secondary consideration. In both these classes, if the surgical conditions be such as require only drainage and ventilation of the sinus, as in nearly all acute and subacute cases, nature has pointed the way of relief to be intranasally, through the nasofrontal duct and the anterior ethmoidal labyrinth. In fact, many of these cases, seen early, yield to therapeutic measures. But if the surgical conditions be such as demand obliteration of the sinus, then the external operation substitutes or supplements the intranasal operation. Thereby the surgical conditions can be dealt with more sanely and safely, and the time of healing greatly shortened, while the deformity is nil or slight in the great majority of cases.

Preceding the external operations, skiagrams should be made showing the exact location and the extent of the sinuses, as they prove of material aid in formulating and expediting operative procedures. When it is impossible to obtain skiagrams, an effort should be made to ascertain the height of the sinuses by catheterizing through the natural passages or by breaking through the agger nasi cell, by a method recently detailed by Mosher,[1] and which will be referred to later.

When the toxemia is severe, a bacteriologic examination of the pus from the sinus should be made, and if streptococci predominate, injections of antistreptococcic serum, antedating the operation, will hasten convalescence. Moreover, some days previous to the external operation communication between the sinus and the nose should be established by an intranasal operation. This procedure may prove not only a preliminary step to, but a preventive measure of the external operation. Mosher,[2] outlining a new method of obliterating the nasofrontal duct by entering the sinus through the agger nasi cell, cites two cases, in one of which this procedure prevented him from doing the Killian operation.

Doubtless all of us have had this experience, so it is useless to dwell on the technic of partial or complete turbinectomy of the middle turbinate and obliteration of the nasofrontal duct and anterior ethmoidal cells, etc. But, in passing, I wish to refer to the location and relation of the agger nasi cell as described by Mosher. He claims that "in a large proportion of subjects there is a distinct swelling on the inner upper surface of the ascending process of the superior maxilla. The mound made by this swelling is just in front of the anterior end of the middle turbinate and often merges with it. This mound is caused by a cell of the anterior group, which has been named from its position the agger nasi cell." Bearing on this point, P. Watson Williams[3] states that, "just as large cells at the summit of the unciform groove may bulge up into the floor of the frontal sinus, forming the frontal bulla, so may cells develop in the unciform lamella, a large cell there forming the cell of the agger nasi, either projecting up so as to mound into the floor of the frontal sinus, or backwards so as to encroach on and block the hiatus semilunaris or unciform groove." My observations are, that while the agger nasi cell may encroach slightly over the margin of the inner upper sur-

face of the ascending process of the superior maxilla, it develops in the anterior portion of the unciform lamella and lies for the most part on a plane posterior and behind the ascending process of the superior maxilla.

External to the agger nasi cell and in line with it above lies the nasal duct. In the Mosher operation to obliterate the nasofrontal duct and catheterize the frontal sinus, care must be exercised with the curette in breaking through the median walls of the ethmoid cells, lest the outer walls are also penetrated and entrance made into the nasal duct, or, if further back, into the orbit through the lacrimal bone.

Technic of External Operation: Make the initial incision in the line of the brow, beginning at the supraorbital notch or foramen, and carry the incision inward and downward until it reaches a point corresponding to the junction of the T-like suture formed by the articulation of the frontal bone with the nasal and superior maxillary bones. This incision may extend outward if the sinus extends to the outer angle of the orbit, or it may extend downward 5 to 8 mm., should it be decided to remove the upper end of the nasal process of the superior maxilla. After retracting the skin, soft tissues and periosteum, in mass, upward over the anterior sinus wall, remove with chisel and forceps a strip of bone 6 to 8 mm. wide from the anterior wall of the sinus, beginning at a point above the supraorbital notch and extending inward and downward until its median margin reaches the nasal process of the superior maxilla at the junction of its articulation with the frontal and nasal bones. Thence continue down the nasal process of the maxilla 5 to 8 mm. only if the emergencies of the case demand it, being careful that the median border of this bone removal does not trespass upon the nasal bone, but follows its articulation with the nasal process of the maxillary bone. To expose the nasal process of the maxilla, after extending the incision through the soft parts along the line above indicated, retract same, with the periosteum, externally to the inner orbital margin. The nasal process having been removed to the extent desired, 5 to 8 mm., preferably with strong biting forceps, we have free and direct access not only to the anterior ethmoidal cells, but to the posterior ethmoidal cells and the sphenoidal sinus, should these latter groups be involved.

However, in a great majority of cases the removal of a strip

of bone 5 to 8 mm. wide from the anterior sinus wall, extending from the supraorbital notch to the nasal process of the superior maxilla, gives direct inspection of the entire sinus and affords ample access to break down all septa and to curette the pyogenic or necrotic mucous membrane of the sinus, as well as the nasofrontal duct and anterior ethmoidal cells, if these latter have not already been obliterated by a preliminary intranasal operation. But if the posterior ethmoidal and sphenoidal sinuses are involved, the partial removal of the nasal process of the maxilla furnishes the needed room for direct inspection and access with curette and forceps to obliterate them with entire safety. In very rare cases it may be feasible to remove a portion of the sinus floor and median orbital wall. The sinus may be lightly packed with a narrow strip of sterile gauze and drainage secured by allowing the ends of this gauze strip to pass below intranasally, and externally, at the lower angle of the external incision, which is sutured save at this point. This gauze should be removed on the second day and an adhesive strip adjusted to the lower angle of the external wound. In suturing the external wound it is important that close coaptation of the periosteum be made throughout, to facilitate granulation beneath. Through and through sutures should be used. Equal care should be taken to accurately approximate the skin margins, with fine sutures, and the line of healing will be imperceptible.

Advantages: (1) The simplicity in this modification of the Killian operation, the author hopes. will appeal to the man of average skill and moderate experience who may be confronted with this important work. Simple technic is safe in itself, and it indirectly shortens the time of operation and anesthesia, thereby lessening shock. (2) With the largest sinuses, reaching to the temporal side of the orbit, by extending the bone removal of the frontal sinus wall 5 to 7 mm. outward from the supraorbital notch, where it began, to the temporal side, direct inspection and access is gained to the remotest recesses, affording easy and complete removal of septa and diseased tissue. And in case the posterior ethmoidal and sphenoidal sinuses are involved, by the extension of the bone removal 5 to 8 mm. down the nasal process of the superior maxilla, we again obtain direct inspection and access from the front and on straight lines, instead of making entrance from the side, as in the Killian

operation. The last condition is no mean advantage when directions mean so much in guiding and limiting us within the bounds of safety in dangerous regions. Moreover, by limiting the median margin of the bone removal to a line corresponding to the articulation of the nasal bone with the nasal process of the maxilla, the danger of injury to the olfactory groove and cribriform plate is minimized. (3) This modification not only conserves the supraorbital ridge, but also the median orbital margin, and leaves the contour and symmetry of the face normal; therefore, the cosmetic effect is ideal. The risk of diplopia is avoided as the pulley of the oblique is not disturbed.

The accompanying photographs of a case where I did a double radical frontal sinus operation, the original Killian and the modified Killian, on the right and left sides, respectively, illustrate the ideal cosmetic effects obtained on both sides, though the right side is slightly flatter at the inner orbital angle, due to the partial removal of the orbital floor.

I regret that the attempt at skiagrams in this case was a failure, as I should like to have illustrated the magnitude of the sinuses, both of which extended beyond the outer orbital angles and contained many septa.

The patient left the hospital on the sixth day, resumed her household duties in two weeks, required practically no after-treatment, and in twelve weeks had gained thirty pounds.

REFERENCES.

1. The Laryngoscope, September, 1911, p. 946.
2. Ibid.
3. Rhinology, p. 24.

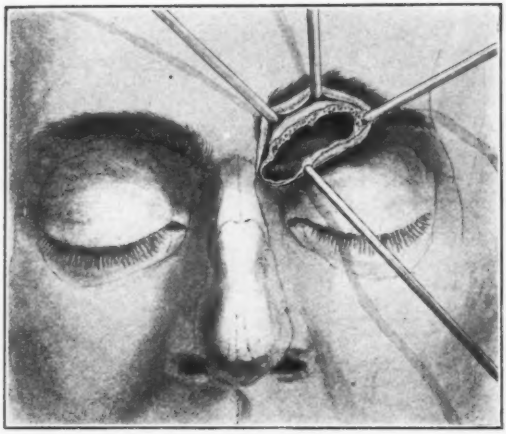

FIGURE I.

This drawing, by light line tracings, shows the articulations of the frontal and nasal bones and the ascending process of the superior maxilla. The incision in the soft tissue is indicated on the right by a heavy line. The extent of the bone removal from the anterior wall of the sinus is indicated on the left.

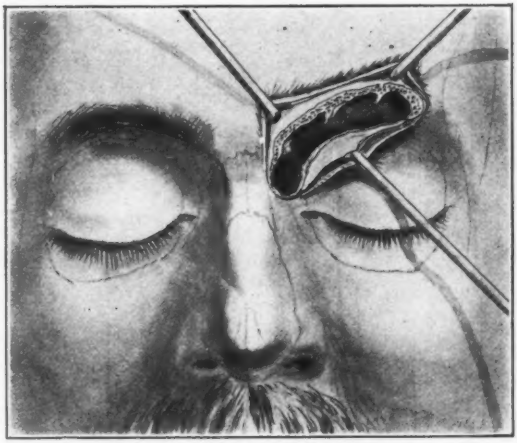

FIGURE II.

This drawing shows the more extensive incision in the soft tissues, also the greater bone removal required when the frontal sinus is very large and other sinuses are involved. By the removal of the upper end of the ascending process of the superior maxilla, direct access, on straight lines, is had to the ethmoid and sphenoid sinuses.

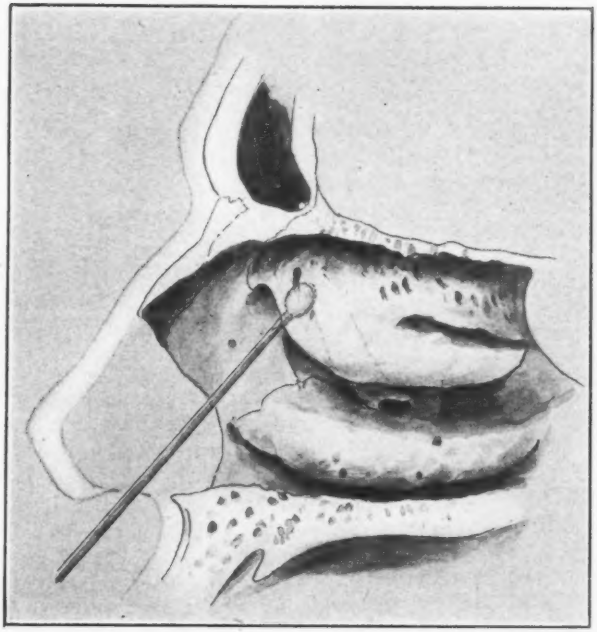

FIGURE III.

This drawing shows the location and relation of the agger nasi cell and Mosher's method of breaking through it with curette to enter the frontal sinus intranasally.

XL.

ON THE ORIGIN OF COMPENSATORY TONUS AFTER DESTRUCTION OF THE LABYRINTH.*

By Geo. E. Shambaugh, M. D.,

Chicago.

In 1909 a young man consulted me because of sudden loss of hearing. He was 27 years old, and had suffered from chronic suppuration in both ears since childhood. Five years previously he had developed an acute mastoiditis on the left side, for the relief of which a mastoid operation had been performed. During this trouble he suffered for a time from vertigo, and since then has not been able to hear with that ear. He came to me five or six days after the sudden loss of hearing in the right ear. For several months previous to this accident he had experienced from time to time attacks of vertigo (fistula). The loss of hearing in the right ear was associated with the development of pain in the ear and an increase in the purulent discharge. When I saw him he was complaining of severe headache and had a temperature of 101° F. The patient stated that at no time after the sudden loss of hearing in the right ear had he experienced any vertigo. An examination by me failed to discover any evidence of spontaneous nystagmus, nor could any response be obtained, either by the rotation or the caloric tests. There was no vestige of hearing in either ear. A diagnosis was made of destruction of the left labyrinth of five years' duration, and of diffuse suppuration of the right labyrinth of one week's duration. Because of the fever and headache, a radical mastoid operation with labyrinth exenteration was at once performed. A fistula was discovered in the horizontal canal, and the labyrinth was found filled with pus. No labyrinth symptoms followed the operation and the patient made a good recovery.

*Read before the American Otological Society, May, 1912.

Voss[1] reports a case where he had made a diagnosis of long standing destruction of one labyrinth[2] and where an accidental opening was made in the labyrinth on the other side during a mastoid operation. There developed in this case a pronounced nystagmus to the opposite side, just as though the accident had occurred in a case where both labyrinths were normal. The spontaneous nystagmus in this case reported by Voss did not disappear completely until after several months.

Experiments on animals, where the second labyrinth is destroyed shortly after the disturbance of equilibrium that follows the loss of the first labyrinth has been readjusted, seem to show the symptoms of disturbed equilibrium which follow the loss of the first labyrinth.

The symptoms observed in these cases are of special interest in connection with the question of the origin of labyrinth tonus, and particularly of the development of the compensatory tonus following the destruction of a labyrinth. To assume the existence of automatic tonus centers, and to refer the explanation of the phenomena of labyrinth tonus to these centers, is hardly a satisfactory answer to these questions, especially since there is no analogy in physiology for the existence of such centers and because all of these phenomena find a satisfactory explanation without these tonus centers.

Normally the voluntary muscles are under the influence of tonus impulses from the labyrinth. These impulses from the two labyrinths are equal, though antagonistic, and a state of equilibrium is sustained. The sudden cessation of tonus impulses from one labyrinth disturbs this equilibrium and a spontaneous nystagmus develops. It is well known that a loss of one labyrinth must take place rapidly in order to produce symptoms of disturbed equilibrium. Where the loss of function in the one labyrinth is gradual, the process may go on to complete destruction of function without at any time giving rise to symptoms of disturbed equilibrium. Moreover, it is known that in those cases where there has been a sudden destruction of function in one labyrinth, the disturbance of equilibrium which is thus occasioned by the complete unilateral suppression of tonus impulses from the labyrinth, disappears after a shorter or longer interval.

The explanation for the readjustment which takes place in the disturbance of equilibrium following the unilateral de-

struction of one labyrinth seems quite clear. This readjustment is due to the development of compensatory tonus impulses to take the place of the tonus from the destroyed labyrinth. When the loss in function is slow enough, the development of these compensatory impulses takes place hand in hand with the loss of the labyrinth tonus, and no disturbance of equilibrium can develop.

Before we can understand the phenomena of labyrinth tonus it is necessary to have in mind certain facts in the physiology of the semicircular canals. These are: first, that the hair cells on one side of a crista are stimulated by an endolymph current in one direction, and the hair cells on the opposite side of the crista are stimulated by a current in the opposite direction; second, the hair cells on one side of a crista, when stimulated, produce nystagmus in the plane of its canal and directed towards one side, while the stimulation of the hair cells on the opposite side of the same crista produce nystagmus in the same plane, but directed to the other side; third, the reaction following the stimulation of the hair cells on the two sides of each crista is unequal; fourth, in each canal the greater reaction follows the stimulation of those hair cells, impulses from which direct the nystagmus towards the same side. Tonus impulses from the labyrinth have their origin in the hair cells of the cristæ. From each labyrinth, therefore, arise tonus impulses for the muscles which direct nystagmus to the same side, as well as for the muscles which direct nystagmus to the opposite side. The stronger tonus impulses from a labyrinth are those which go to the muscles directing nystagmus to the same side, because, as just pointed out, the stimulation of the hair cells which give rise to these impulses produce a greater response than does the stimulation of those hair cells, impulses from which go to the muscles directing nystagmus to the opposite side. When the tonus impulses from one labyrinth are suddenly suppressed the balance of equilibrium is disturbed, and the tonus from the normal labyrinth, acting without the restraint of impulses from the opposite side, produce nystagmus directed towards the normal side.

What now is the origin of these compensatory tonus impulses that develop after the destruction of a labyrinth and which restore the disturbed balance in equilibrium? These

compensatory impulses may have two sources. One source is the compensatory increase in those tonus impulses from the remaining labyrinth which direct nystagmus to the opposite side. Should restoration of equilibrium be due entirely to compensatory tonus from this source, it would mean that the tonus impulses from the hair cells on the two sides of each crista in the remaining labyrinth are equal. This in turn should mean that a current of endolymph in one direction in a canal would produce a reaction just as great as a current in the opposite direction. This phenomena has been observed by Ruttin[3] in several cases where there had been a long standing destruction of one labyrinth. Here he observed that the nystagmus following rotation in one direction was just as great as that following rotation in the opposite direction In cases of this sort, where the restoration of balance after the destruction of one labyrinth is due entirely to compensatory impulses from the opposite labyrinth, it is quite clear that in the event of the sudden destruction of the remaining labyrinth, no pronounced disturbance of equilibrium and no nystagmus should follow, for the reason that the destruction of the second labyrinth would be the same as the simultaneous destruction of both labyrinths in its effect on tonus. It is possible that in the case reported in this article the absence of disturbed equilibrium after the loss of the second labyrinth is to be explained in this way.

In most of the cases where a restoration of equilibrium has been accomplished after the loss of one labyrinth, it is evident that this is not due entirely to the compensatory increase in tonus from the opposite labyrinth, because in applying the rotation test in these cases the afternystagmus to the normal side is found to be much stronger than to the defective side. This test shows that the reaction following stimulation of the hair cells on the two sides of the crista is still unequal. It is clear, therefore, that in most cases the restoration of equilibrium after a unilateral labyrinth destruction is accomplished by a compensatory tonus which, in part at least, is developed independent of the opposite labyrinth. It is even possible that in some cases the restoration of equilibrium is accomplished entirely through the development of this extralabyrinth compensatory tonus. I cannot accept the view that there exist automatic tonus centers in Deiter's nucleus, where

tonus impulses are stored or originated. The tonus impulses which emanate from Deiter's nucleus have their origin in afferent impulses from the labyrinth and elsewhere. These impulses are in a measure modified in their passage through Deiter's nucleus, but tonus impulses are neither stored nor developed automatically here.

It is known that afferent impulses other than those from the labyrinth convey tonus to the voluntary muscles. The tonus impulses from the labyrinth are probably the more important, as they are the more delicate. The extralabyrinth tonus impulses are the cruder and more primtiive. Those from the labyrinth come from a highly specialized mechanism developed for the particular purpose of preserving equilibrium. A sudden loss of the labyrinth destroys not only the labyrinth tonus from this side, but the sudden shock to the center in Deiter's nucleus destroys temporarily much of the extralabyrinth tonus. This it does by a process which Monakow[4] has designated as diaschisis. The rapid recovery of the disturbed equilibrium following the loss of a labyrinth is to be explained through the action of the extralabyrinth tonus. The first process in the recovery of equilibrium is the restoration of the normal extralabyrinth tonus through the disappearance of the suppression of this tonus by the action of diaschisis. But this restoration of the normal extralabyrinth tonus is not sufficient to restore the disturbed equilibrium arising from the unilateral loss of labyrinth tonus. Its place must be supplied by the development of an additinal tonus. This additional compensatory tonus, which early takes the place of the tonus from the destroyed labyrinth, seems to develop chiefly from the extralabyrinth impulses. This is shown by the fact that the response of the hair cells on the two sides of the cristæ in the remaining labyrinth continues to be unequal. In applying the rotation test the afternystagmus to the normal side is greater than to the opposite side. It appears to be only in cases of long standing destruction of one labyrinth where the compensatory tonus from the remaining labyrinth supplants completely this compensatory extralabyrinth tonus. In these cases the response of the hair cells on the two sides of the cristæ in the remaining labyrinth is the same, and in the rotary test the afternystagmus to the defective side is as strong as towards the normal side.

It is evident that these extralabyrinth compensatory tonus impulses which develop after the destruction of one labyrinth will tend to balance the impulses from the several cristæ of the remaining labyrinth. In case of the sudden destruction, therefore, of the second labyrinth, a disturbance of equilibrium and a nystagmus should develop exactly as though the function of the first labyrinth had been restored. The nystagmus will be directed to this side. The disturbed equilibrium and spontaneous nystagmus which follows the sudden destruction of the second labyrinth are produced by the compensatory extralabyrinth tonus, which had developed to take the place of the tonus from the labyrinth first destroyed, acting now without the restraint of tonus impulses from the labyrinth last destroyed. The restoration of equilibrium and disappearance of the spontaneous nystagmus which follows the destruction of the second labyrinth is dependent entirely on the development of extralabyrinth tonus to take the place of the tonus from this labyrinth.

The restoration of equilibrium which follows the destruction of both labyrinths is never a complete restoration of the normal equilibrium. The extralabyrinth tonus which supplants the labyrinth tonus is not as perfect a mechanism for preserving the equilibrium as is the tonus from the highly specialized sense organs in the labyrinth. In the case of the sudden loss of one labyrinth, the rapid adjustment of equilibrium which follows is dependent, as pointed out above, on the development of the extralabyrinth compensatory tonus. The readjustment is not a complete substitute for the labyrinth tonus. It is only in cases of long standing unilateral labyrinth destruction where the restoration of equilibrium is apparently perfect, and here the compensatory tonus from the opposite labyrinth has fully developed so as to supplant completely the compensatory extralabyrinth tonus.

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XLI.

SYPHILIS OF THE INNER EAR.*

By Otto Glogau, M. D.,

New York.

Ever since syphilis began to spread over the world, its aural symptoms have been known. A scientific study of syphilitic ear lesions, however, was possible only after modern otology, guided by clinical and pathologic researches, offered the ways and means of systematic examination of the inner ear, which has revealed hitherto unrecognized conditions.

As to how many pathologic changes of the inner and middle ear, now classified as autochthonous conditions, are really due to syphilis as the underlying cause, it is as yet not possible to state. There is also a lack of thorough investigation and accurate statistics of inner ear complications in the different stages of clinically diagnosed syphilis.

With this idea in mind the writer undertook to examine, for a certain length of time, by all advanced methods available, the function of the inner ear of all syphilitic patients of various skin departments, regardless of the fact that there were no aural symptoms. At the same time, the writer, in his clinics and private practice, scrutinized systematically all cases of inner ear involvement. The previous history was studied, the skin and the natural orifices, especially the nose and throat, were carefully examined for the purpose of determining the presence of syphilis, and the Wassermann test was also applied. In doubtful cases antisyphilitic treatment was administered to corroborate the diagnosis ex juvantibus et nocentibus. The writer has not yet wholly finished his investigations and withholds the final results of the same for a more detailed communication. This report, therefore, is to be considered as a preliminary one. A thorough review of the respective literature with a critical study of the clinical data laid down by

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the different writers and special reference to the pathology will be given first. The more important and rare types of syphilitic infection of the inner ear selected from the writer's own cases will be added.

The organ of hearing may be affected by all possible syphilitic tissue changes: of the skin on the external ear, of the mucous lining in the midle ear, of the periosteum, bone and nerve in the inner ear. It may be considered as a rule from which there exist not a few exceptions, that the external ear and the pharyngeal opening of the eustachian tube is the seat of the primary lesion, while the middle ear is affected by secondary and the inner ear by tertiary syphilitic tissue changes. In describing the symptoms and pathology of syphilitic labyrinthitis we have to differentiate between hereditary and acquired syphilis as causative factors. As the inner ear, on account of its close anatomic relationship to the middle ear, is affected as a rule simultaneously with or secondarily to the latter, the syphilitic changes of the middle ear will have to be mentioned first.

In the secondary stage of acquired syphilis the middle ear may be affected primarily or by extension from the nasopharynx and external canal, where papules on the drum (case of Lang) may cause a perforation and obstinate middle ear suppuration. Gerber, in his excellent monograph on syphilis of the Nose, Throat and Ear, is of the opinion that most syphilitic affections of the middle ear are secondary to syphilitic changes in the epipharynx and mesopharynx. Gradenigo, in Schwartze's Handbuch, goes even further and claims that syphilitic changes in the inner ear occur only secondary to pathologic changes (hyperemia, slight disturbances of nutrition, etc.), due to acute or chronic middle ear inflammation. He believes, too, that secondary luetic affections of the middle ear are sequelæ of syphilitic alterations in the nose and throat.

It is very important to note that auricular symptoms, like tinnitus, impairment of hearing, and otalgias, may be due to syphilitic changes of the throat and nasopharynx exclusively, while the middle ear itself may still be in a normal condition. There is no method of differentiating clinically between specific and nonspecific middle ear catarrh, as the same symptoms may prevail. Schwartze lays stress upon the exacerbating

temporal headaches, rapid impairment of hearing and shortening of bone conduction, the latter symptom pointing to a syphilitic infection of the inner ear. The presence of an amber colored exudate, coming from the middle ear after paracentesis (Moos, Kirchner, Schwartze), and the tendency to complications like caries and necrosis of the mastoid bone, sinus thrombosis, facial paralysis and cerebral complications (Wilks, Leschevin, Neumann, Bright, Habermann, Buck) are mentioned. The most important diagnostic points, however, are the rapid impairment of hearing and the simultaneous affection of the labyrinth. The presence of the spirocheta within the exudate and a positive Wassermann reaction will corroborate the diagnosis. The pathologic changes of the mucous lining of the middle ear are similar to the secondary syphilitic changes of the nasopharyngeal mucous lining.

The syphilitic changes of the inner ear proper (vestibulum, cochlea and semicircular canals) occur mostly at the end of the secondary and at the beginning of the tertiary syphilitic stage. They are either combined with catarrhal or suppurative middle ear inflammation or present as a primary symptom. Politzer noticed labyrinthine symptoms as early as seven days after the appearance of the secondary rash, while most observers (Gerber, Schwartze, Rosa, Habermann) observed their appearance from three to seven months after the infection.

It is somewhat dogmatic to differentiate in vivo between secondary and tertiary labyrinthitis, as no clinical symptom is characteristic for the pathologic changes of either one. For this reason Gradenigo bases his classification of syphilitic labyrinthitis on clinical observations exclusively and establishes the following groups: (1) Labyrinthitis with slow progress, (2) with rapid progress, (3) with apoplectiform onset.

Gerber recognizes distinctly a secondary syphilitic labyrinthitis occurring about six months after the infection and being in most instances accompanied or preceded by secondary syphilitic changes of the nose and throat. The pathologic changes consist of irritative inflammation, hyperemia (Toynbee), and small cell infiltration (Moos). There are no histologic examinations at hand.

The clinical symptoms depend upon whether we have to deal with an isolated or simultaneous involvement of the

acoustic and static apparatuses. Thus impairment of hearing, shortening of bone conduction, subjective noises, dizziness and disturbances of equilibrium may be present, while Rinne's test usually remains positive. In this connection it may be mentioned that Jung believes the acoustic nerve proper to be exclusively involved in the secondary syphilitic stage; most of the writers, however, believe this to occur in the tertiary stage.

Tertiary syphilitic labyrinthitis is met with in about 7 per cent of all cases of internal otitis (Gradenigo, Brueckner, Gerber) and may occur from one to thirty years after the infection. The pathologic changes, as demonstrated by microscopic findings, are as follows: Ankylosis of the stapes, thickening of the vestibular periosteum, marked injection of the cochlea with similar changes in the middle ear (Toynbee); small cell infiltration and hyperplasia of the connective tissue between membranous and osseous labyrinth, infiltration of Corti's organ, of the ampullæ and of the membranous semicircular canals (Moos); hyperostosis of the footplate of the stapes (Schwartze); atrophy and decay of the cells within the ganglion spirale (Politzer, Moos, Steinbruegge).

As in nearly all of the above mentioned reports tertiary syphilitic changes in the middle ear have been met with, a short description of the latter is justified. The drum membrane is usually normal; gummata on its internal and external surface (Ravogli), and changes similar to those of parenchymatous keratitis (Habermann), have been described. Gummata at the pharyngeal opening of the eustachian tube have been frequently met with and are included by Gerber in his symptom complex of lues occulta cavi pharyngonasalis.

The pathologic changes within the middle ear proper consist of atypical perivascular small cell infiltration; as sequelæ thereof we find alterations of the blood vessels, formation of new connective tissue, periosteum and bone, and destruction of fundamental tissues. Clinically the following three processes may be diagnosed: (1) Serous catarrhs with frequent relapses; (2) suppuration with more or less pronounced caries of the bone; and (3) sclerosis. The frequency of positive Wassermann reaction in both otosclerosis and progressive nerve deafness (in the former 76 per cent, in the latter 52 per cent, according to Buck), suggests that in most cases of

this kind syphilis is the underlying condition. The cardinal symptoms are: Rapid impairment of hearing, considerable shortening of bone conduction, subjective noises, nightly headaches, otalgias, and especially symptoms pointing to an involvement of the labyrinth and acoustic nerve.

In tertiary labyrinthitis the following symptoms present themselves: Impairment of hearing or deafness, subjective noises, dizziness, frequently combined with nausea and vomiting, disturbances of equilibrium, shortening of bone conduction, lowered perception of the tone of the high tuning fork. Rinne's test is usually positive, and Weber, in unilateral affection, pointing to the healthy side. The onset is frequently acute, resembling Meniere's symptom complex; this represents Gradenigo's above mentioned apoplectiform type, of which thrombosis following syphilitic endarteritis is the supposed underlying pathologic condition. A positive Wassermann, in absence of other syphilitic symptoms, will confirm the diagnosis. Politzer, however, bases his diagnosis in the young adult with normal middle ears on the rapid impairment of hearing, marked shortening of bone conduction and lowered perception of high tones.

The acoustic nerve may be affected by any of the following syphilitic processes at the base of the brain or within the pyramid bone (Gerber): (1) primary gummatous neuritis, in the early syphilitic stage; (2) gummata and periostitis of the pyramid bone; (3) basal gummatous meningitis; (4) chronic inflammation of the dura (paralysis by compression); (5) syphilitic affection of the outer cranial periosteum (Rosenstein). The four conditions mentioned last belong to the tertiary syphilitic stage.

Early symptoms are: Acute onset with dizziness, vomiting, deafness, combined or followed by facial paralysis. Of the greatest diagnostic importance is Gradenigo's symptom of shortening of the perception of the tones of the middle tuning forks and of functional exhaustibility. In the majority of cases, however, it is difficult to differentiate the syphilitic affection of the acoustic nerve proper from those of the labyrinth and of the brain. Where gummatous basal meningitis is the underlying condition the symptoms of the latter will prevail. Thus, besides the general symptoms of vomiting and headaches, and the acoustic symptoms of deafness, noises and

acoustic hyperesthesia, changes of the optic, oculomotor, trigeminus, abducens and facial nerves will be present.

It is a well known fact that the acoustic nerve may atrophy in syphilitic manifestations of locomotor ataxia. Habermann gives the following description of the respective microscopic changes: Almost perfect atrophy of the cochlear and partial atrophy of the vestibular branches of the acoustic nerve. Replacement of the nerve fibers by connective tissue, into which there were imbedded numerous corpora amylacea. Neuritis of the ganglion cells within Rosenthal's canal. According to Gradenigo, the pathologic changes occurring at apoplectiform tabetic deafness are hemorrhages within the nuclei of the acoustic trunk, due to specific endarteritis.

In congenital syphilis the inner ear is more frequently and more severely affected than in the acquired form. The symptoms do not differ very much; they are, however, more pronounced and less amenable to treatment. Congenital syphilitic deafness was first described by Hutchinson as one of the three cardinal symptoms of lues hereditaria tarda, the two others being interstitial keratitis and deformation of the teeth. The affection of the inner ear, being mostly bilateral, may set in very early, but occurs usually at the age from ten to thirty; it is met with two or three times as often in women as in men, where it appears clinically as sclerosis of the middle and inner ear. When labyrinthine lues sets in in early childhood, it mostly leads to deafmutism; the same condition is brought about when the syphilitic inner ear changes occur in the fetus. Moos, Steinbruegge and Panse found in the temporal bones of syphilitic deafmutes, chronic suppuration and caries in both the middle and inner ear. Otto Mayer examined the temporal bones of eleven luetic children of the age between ten days and seventeen months, and came to the following conclusion: Specific meningitis in hereditary luetic children brings about a specific interstitial inflammation of the acoustic nerve that progresses towards the inner ear with different degrees of intensity.

Hutchinson and Hermet state that deafness in late hereditary syphilis is due to syphilitic neuritis of the acoustic nerve. Kipp and Fournier believe a gummatous process on the floor of the fourth ventricle to be the underlying condition. On the other hand, such pathologic findings as hyperemia of the

vestibule, hemorrhages and destructive changes within Corti's organ, point to a primary labyrinthitis, while periostitis, bone formation and syphilitic processes within the middle ear prove that in many cases the tympanum must be considered as the primary focus. Gradenigo lays stress upon the incidental involvement of the middle ear and the nasopharynx; he also believes that the pathologic changes within the inner ear are similar to those occurring in cornea, iris and choroid.

The following symptoms are characteristic: Sudden impairment of hearing, mostly bilateral, rapidly progressing to deafness, marked shortening of bone conduction, reduced perception of high tones, excruciating subjective noises of long duration, dizziness, disturbance of equilibrium and sometimes facial paralysis. The drum is normal or slightly clouded, hyperemic or thickened. Specific ocular symptoms, as interstitial keratitis, iritis, iridochoroiditis, retinitis, neuroretinitis and other specific somatic changes are usually present, especially those of the nose, throat and nasopharynx. The prognosis of all syphilitic ear affections, except those in the earliest stage, is bad. The treatment, being that of general syphilis, will not be considered in this communication. The writer wishes to report the following cases:

Case 1.—Mrs. B., age 55 years, progressive impairment of hearing in both ears for nearly five years. At intervals of a few days she suffers from attacks of dizziness, combined with nausea, falls down and loses consciousness for short time. She experiences excruciating pains and head noises. Positive specific history.

Present state. Through both drums there is noticeable a red discoloration of the promontory, which is considered a characteristic symptom of otosclerosis. Perception for high and low tuning forks markedly reduced, pronounced shortening of bone conduction, extreme irritability of the static apparatus upon rotation and injection of water. Rinne negative, Weber indistinct. Deviation of septum to the left, hypertrophy of the middle turbinate on the right side. Diagnosis: Tertiary syphilitic involvement of both the middle and inner ears.

This case is different from any of Gradenigo's types, as its slow progress appears interrupted by apoplectiform attacks. The presence of nasal obstruction corroborates the above mentioned theory, that syphilitic lesions of the middle and inner

ear occur mostly when predisposing nasopharyngeal factors are present. Antisyphilitic treatment stopped the attacks, without influencing the other aural symptoms.

Case 2.—Dr. L., referred to me by Dr. Dietrich. Patient is 40 years of age and acquired lues six weeks ago. With the appearance of the secondary rash, two days ago, patient became suddenly deaf in the right ear, while suffering an attack of dizziness combined with vomiting and loss of consciousness. He was two days in bed and now stumbles when standing or walking. His mentality is lowered since the attack, the memory nearly gone. He was suffering from chronic catarrh of the right middle ear.

Present state: Left drum, middle and inner ear normal. Right side, drum retracted, dull. Reduced perception for the low tuning fork, no perception for high tones. Bone conduction almost lacking. Rinne positive, Weber towards the left side. Conversation and whisper voice not heard. No changes in the optic nerves. Posterior tip of right inferior turbinate hypertrophied. Diagnosis: Secondary syphilitic labyrinthitis with apoplectiform onset.

The remarkable feature of this case is the apoplectiform onset, involving the inner ear six weeks after the infection. Here, too, the nasopharyngeal condition must be considered as a predisposing factor. Antisyphilitic treatment was of no avail.

Case 3.—Mrs. L., age 38 years. Ten years ago was infected by husband. Was treated by inunctions and injections. Two years ago she complained of headaches and fullness in the ear. Catheterization did not improve the condition. On account of her pains in the throat, tonsillectomy was performed. Two weeks ago she experienced throbbing headaches, especially around the temples, subjective noises, fullness in both ears, especially the right one, and pains radiating into the throat.

Present state: Conversation and whisper voice, low and middle tuning forks on both ears normally perceived. Lowered perception for the high tuning forks and very marked shortening of bone conduction, both more pronounced on the right side. Rinne positive, Weber indistinct. The function of the vestibular apparatus, upon rotation and injection of water, proves to be normal. Lingual tonsil enlarged; nose and

throat in catarrhal condition. Diagnosis: Tertiary syphilis of the acoustic apparatus of the inner ear.

This case represents Gradenigo's slowly progressing type; here, too, we find predisposing nasopharyngeal conditions.

Case 4.—Mrs. C., age 35 years. Referred to me by Dr. Adolph Schoen. She acquired syphilis twelve years ago. She has had foul discharge from the nose for one year. One week ago she had an attack of dizziness combined with vomiting; immediately afterwards she noticed that the left side of her face was somewhat paralyzed and that the hearing of her left ear was rather impaired.

Present state: Large luetic perforation of septum, much necrotic bone present. Left posterior tip of inferior turbinate extremely hypertrophied. The left facial nerve is slightly paralyzed. The functions of the right ear are normal. On the left ear the tones of the low and high tuning forks are normally perceived. The tones of the middle pitched tuning forks, however, are not perceived at all. A remarkable feature is the functional exhaustibility of the nerve; when tried in succession, the same tuning fork tests give different results. This symptom, together with the lowered perception of the tone of the middle pitched tuning fork, is, according to Gradenigo, pathognomonic for affection of the acoustic nerve. Diagnosis: Tertiary syphilitic affection of the acoustic nerve.

Upon injection of salvarsan, the slight remnants of facial paralysis disappeared and the discharge from the nose stopped. The acoustic nerve recovered somewhat; when last seen the patient could perceive the middle tones almost normally. The functional exhaustibility, however, persisted.

Case 5.—R. P., a negro, age 32 years. Patient of my class at the North-Western Dispensary. He acquired syphilis four months ago. For the past few days he was dizzy, and had unpleasant sensations within the left ear. The acoustic apparatus is normal on both sides. The function of the right static apparatus is normal, too. The left static apparatus, however, proves to be very irritable. Violent nystagmus with dizziness and nausea occur rapidly upon injection of both cold and warm water and after turning the patient a few times on the rotation chair. Diagnosis: Secondary syphilitic affection of the left vestibular apparatus.

Case 6.—M. O., girl age 14 years. Was born with a saddle

nose. At the age of 3 years, on account of nasal obstruction, she had a nasal operation performed. In her fifth year impairment of hearing was noticed, which soon led on to deafness. Father gives a luetic history.

Present state: Deviation of septum. Loud conversation voice perceived ad concham. Marked shortening of bone conduction, no perception of high tones. No vestibular abnormalities. Pathognomonic eye symptoms lacking, Hutchinson's teeth present. Diagnosis: Late hereditary syphilis of the acoustic apparatus of the inner ear.

In conclusion, the writer would like to state: (1) In all cases of middle and inner ear affection the tuning fork tests should be applied both in dispensary and private practice. Thus in many instances syphilitic aural affections may be diagnosed. (2) All cases of involvement of the labyrinth and of the middle ear, especially of otosclerosis, should be scrutinized for syphilis, by investigating the previous history, inspecting the skin and orifices and by applying the Wassermann test. (3) Early antisyphilitic treatment may in some instances restore the function of hearing and equilibrium, while in overlooked cases catheterization and other routine procedures hasten the destructive process.

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XLII.

THE PATHOLOGY OF LABYRINTHITIS.*

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No subject in otology has provoked so much discussion in recent years as that of the indications for surgical interference in diseases of the labyrinth. It is not my purpose at the present time to discuss the contentions of various authors nor to consider here the literature on this subject. I will refer to the indications—the result of several years of personal work—only to seek their underlying principles.

Two fundamentals guide me in these indications:

1. Not to destroy a still functionating labyrinth. Because we should take due heed before we destroy an organ of sense. And because there still remains time for surgical interference, when there is an advance of the disease to the stage of dangerous diffuse suppuration, as shown by complete loss of function.

2. The simple surgical principle, ubi pus ibi evacua. That is, as soon as I have made a diagnosis of diffuse suppurative labyrinthits, I do not content myself with the radical operation, but also open the labyrinth. For what surgeon will be satisfied in opening the superficial of two collections of pus (middle ear and labyrinth), which have but indifferent communication, and leave the care of the deeper abscess to Mother Nature?

The difficulties which sometimes present themselves in summing up the indications lie in the diagnosis. In an inflammatory process, such as labyrinthitis is, the stages and grades

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from one degree to another are manifold. Hence the sharp distinctions which we must assume for the sake of nomenclature are in a way unnatural. Our table of indications can on this account only contain types. And the weakest and most vital point must naturally be serous labyrinthitis, since this disease marks the transition from circumscribed to diffuse suppurative labyrinthitis. This transition is only too often not sharply defined.

We can only deal here with those inflammatory diseases of the labyrinth which arise consecutively from purulent otitis media. If such a process attacks only part of the labyrinth, we call it circumscribed labyrinthitis. We do not attempt to distinguish between nonsuppurative (serous) and suppurative circumscribed labyrinthitis, because this differential diagnosis clinically is as yet impossible. We also include simple perforation of the labyrinth wall (fistula in labyrinth) with circumscribed labyrinthitis; although we know there are cases in which the fistula has occurred without subsequent infection of the labyrinth. (Fig. 1.)

If, on the contrary, the inflammation has spread over the entire labyrinth, we designate this clinical picture diffuse labyrinthitis. In most cases we are able to distinguish clinically between diffuse nonsuppurative (serous) and diffuse suppurative labyrinthitis. (Figs. 2, 3, 4.) Diffuse serous inflammation is always an acute disease with sudden onset. It can, indeed, leave permanent changes in the labyrinth, but these residua are never able to produce a fresh exacerbation of the same process. On this account we cannot include a chronic form of serous labyrinthitis in our classification. (Figs. 4, 5.)

On the other hand, we must classify from another point of view two kinds of serous labyrinthitis. One, which follows circumscribed labyrinthitis, we call diffuse serous secondary labyrinthitis; and one, which arises suddenly and uncomplicated in cases where the labyrinth wall is intact (or at least not perforated), we designate diffuse serous induced labyrinthitis.

On the contrary, in diffuse purulent labyrinthitis we are able to differentiate between acute and chronic forms. In the acute form, which we call the "manifest," the whole labyrinth is filled with a fresh purulent exudate. (Figs. 6, 7.) In the

chronic or "latent" type, the labyrinth is filled, according to the duration of the disease, with the organized products of a suppurative inflammation (granulations, inflammatory tissue, bone tissue). And frequently there are also, at one or another place, products of disease (pus, bacteria) which may later lead to an exacerbation or further extension of the process.

We have, therefore, the following more or less well defined clinical pictures for classification:

1. Circumscribed labyrinthitis.
2. Diffuse serous secondary labyrinthitis.
3. Diffuse serous induced labyrinthitis.
4. Diffuse suppurative manifest labyrinthitis.
5. Diffuse suppurative latent labyrinthitis.

Let us look at these clinical pictures from a closer point of view. To establish the diagnosis we must examine the previous history of the patient for the presence or absence of the socalled vestibular symptoms (vertigo, vomiting, disturbance of equilibrium, nystagmus). We must also in our examination determine the status of the cochlear and vestibular function (degree of hearing, and reaction to the caloric, turning, and fistula tests).

I. CIRCUMSCRIBED LABYRINTHITIS.

The history is usually characteristic. The patient complains of attacks of dizziness—especially after quick movements of the head, in stooping, or when cycling, indulging in athletics, or jumping off and on the street cars, etc. Examination may show vestibular symptoms or not, depending upon whether the patient presents himself during an attack or some time afterwards. The nystagmus may be directed towards the diseased or to the sound side, or even to both sides—despite the theory that each labyrinth upon stimulation produces nystagmus to the same or to the opposite side. Hearing is always present, although it may be very much impaired. As a rule, the hearing distance for loud speech is one-half to one meter, although I have seen cases that could hear at eight meters. The caloric and turning reactions are obtained, usually quite promptly, and the fistula symptom is demonstrable. Of course, there must be cases of circumscribed labyrinthitis in which the fis-

tula symptom cannot be obtained, where there is absolutely no fistula, but such cases cannot be diagnosed with any certainty.

II. DIFFUSE SEROUS SECONDARY LABYRINTHITIS.

The history of these cases is naturally identical with that of circumscribed labyrinthitis, since the former disease always follows the latter. Different, however, are the present symptoms. These are typical of a sudden unilateral arrest of labyrinth function. To be sure, the labyrinth is not destroyed or its function wholly lost. Yet the sudden onset of diffuse inflammation so interferes with the function of the diseased side that a preponderance of the sound labyrinth results—just as in sudden destruction of one labyrinth. Vertigo, vomiting or nausea, disturbance of equilibrium, and rotatory nystagmus to the same side, are the usual symptoms. Naturally the severity of these symptoms varies greatly. Diffuse serous secondary labyrinthitis may arise in cases with circumscribed labyrinthitis of itself, or may follow the inflammatory reaction produced by the radical mastoid operation. In the latter case the rule is that the third to fifth day after operation is the time to await this complication (the onset of labyrinthitis). The function of the labyrinth is usually very essentially impaired—the cochlear more severely than the vestibular.* The hearing distance is very noticeably shortened—even to complete deafness in the diseased ear. The caloric, the turning, and reaction to the fistula test are essentially altered, or may be wholly arrested. If the hearing power, the caloric and turning reactions, and the fistula symptom are abolished, then, of course, we can no longer distinguish between such a serous labyrinthitis and a diffuse suppuration of the labyrinth. This, however, is of no great consequence, since this, the severest type of serous inflammation, probably indicates the transition into suppuraiton.

Always with less severe degrees of serous inflammation, one or the other of the functions of the labyrinth is present. Loss of function is customarily in the following order: Hearing, caloric reaction, turning reaction, fistula symptom. So,

*The explanation for this fact I have found in that the functional element of the cochlea, the perilymph, lies next to the labyrinth wall and is disturbed earlier and more severely than the endolymph, the functional element of the vestibular apparatus.

corresponding to the severity, the following degrees may be given:

1. Hearing, Caloric reaction, Turning reaction, Fistula symptom, } present.
2. Hearing, } absent.
   Caloric reaction, Turning reaction, Fistula symptom, } present.
3. Hearing, Caloric reaction, } absent.
   Turning reaction, Fistula symptom, } present.
4. Hearing, Caloric reaction, Turning reaction, } absent.
   Fistula symptom, } present.

The fifth degree, namely, loss of all function, is, as stated above, impossible to differentiate from suppurative labyrinthitis. If in a given case of circumscribed labyrinthitis, symptoms of diffuse labyrinthitis suddenly arise, the diagnosis of diffuse serous secondary labyrinthitis depends upon the demonstration of at least one (or more) functions of the labyrinth.

DIFFUSE SEROUS INDUCED LABYRINTHITIS.

In one class of cases inflammation extends to the labyrinth through the intact or at least imperforated labyrinth wall. In other cases there is a simple inflammatory edema in the labyrinth. This occurs when the middle ear inflammation is very severe and when the anatomic relations are such that the labyrinth lies in the zone of inflammation. (This is relatively more frequent in acute than in chronic otitis media.) We may also have a "postoperative labyrinthitis," if the inflammatory reaction after the radical mastoid operation extends into the labyrinth.

It goes without saying that the above cases give no previous history of labyrinth symptoms, but only those of an acute or chronic middle ear suppuration. The symptoms of the disease are naturally identical with those of diffuse serous secondary labyrinthitis, but are usually more pronounced. And, too, vertigo usually persists very long, for the onset of the disease is sudden and the patient has had no previous attacks of dizziness. It has been demonstrated time and again in patients who have had one or more attacks of vestibular vertigo, that subsequent attacks are subjectively of much shorter duration. This is easily recognized in, for example, cases of circumscribed labyrinthitis, who have often had attacks of vertigo. These patients experience only the slightest feeling of dizziness when we produce rather decided nystagmus by means of the caloric or turning tests.

Loss of function is in the same order as in diffuse serous secondary labyrinthitis—except, of course, that the fistula symptom is never demonstrable. Because of the latter, diffuse serous induced resembles diffuse suppurative more than diffuse serous secondary labyrinthitis. When hearing is gone and the caloric and turning reactions cannot be obtained, we may be dealing with a diffuse serous induced or with a diffuse purulent labyrinthitis. Fortunately, function is but seldom fully lost in serous labyrinthitis. (Most usually this is caused by an acute otitis.) Moreover, it is certainly of no great consequence if such a case of labyrinthitis is treated as a suppurative one. For this is scarcely other than the most severe type of serous labyrinthitis and also marks the transition into purulent inflammation. (Figs. 2, 3.)

DIFFUSE SUPPURATIVE MANIFEST LABYRINTHITIS.

Here we have to deal with a purulent inflammation that extends in a short time over the entire labyrinth. Loss of labyrinth function is sudden and complete. As the disease may occur in cases who have or have not had circumscribed or serous labyrinthitis, there may or may not be a history of vestibular symptoms. The symptoms of diffuse suppurative manifest labyrinthitis are: vertigo, vomiting, disturbance of equilibrium, and marked rotatory nystagmus to the sound side. The function of the labyrinth is fully destroyed. The patient is deaf and absolutely does not react to the caloric and turning

stimuli (when the latter are unmistakably provable). The fistula test is, of course, negative, even though a fistula exists.

DIFFUSE SUPPURATIVE LATENT LABYRINTHITIS.

As a rule there is a history of vestibular symptoms, especially vertigo. These symptoms may have occurred weeks, months or years before. In rare cases these symptoms are denied, usually because the patient has forgotten. Still there occur isolated cases with "symptomless destruction," in which prominent labyrinth symptoms have never arisen. This I have observed with my own eyes in one case of labyrinthitis. Customarily the labyrinth infection has occurred some time in the past, and we know by experience vestibular symptoms last from three to fourteen days after sudden total destruction of the labyrinth. So, naturally, in cases of diffuse suppurative latent labyrinthitis, no further symptoms are found until the time of the examination.

This labyrinth disease can only be discovered through functional tests. Then we find total deafness, and no reaction to the caloric or turning stimuli, on the diseased side, and the fistula test is, of course, negative. In certain cases there may be an apparent reaction to the turning stimulus. This semblance I have described under the name "compensation." This compensation (that is, the equalness of duration of turning nystagmus for both sides, in spite of an unilateral destruction of the labyrinth), according to my experience, occurs when a total destruction of the labyrinth (bone necrosis, sequestrum formation) exists for a long while.

INDICATIONS FOR THE RADICAL AND LABYRINTH OPERATIONS IN INFLAMMATORY LABYRINTH DISEASE.

The question as to which inflammatory disease of the labyrinth necessitates operative opening of the diseased labyrinth is easily answered according to surgical principles, namely, that every suppurative labyrinthitis requires wide opening and drainage. As we can diagnose the disease only after complete loss of function, so in the same manner we must undertake the labyrinth operation upon complete loss of labyrinth function, namely, in diffuse suppurative, manifest and latent labyrinthitis. We cannot deny that in some circumstances diffuse suppurative labyrinthitis can heal. However, the pa-

tient thus afflicted has the sword of Damocles (meningitis) hanging over his head until this questionable and difficultly controlled healing takes place. And it is just like cutting the thread that holds the sword to do the radical operation in this sort of case and leave the labyrinth to take care of itself.

Then there is the question why we perform only the radical mastoid operation in circumscript, and the two forms of serous, labyrinthitis (cases 1 to 3 in the table). We have many excellent reasons for this. Chiefly because the labyrinth is not fully destroyed, its function partly retained, and the partial impairment itself need not be lasting. Secondly, circumscribed and diffuse serous labyrinthitis heal relatively very often after the primary focus of pus is removed. Then, too, only very exceptionally does an intracranial complication of labyrinth origin occur directly from circumscribed or diffuse serous labyrinthitis, without these labyrinth diseases first going through the stage of diffuse suppuration. Therefore, with the extension of the process, we have in the progressive loss of function the indication for a still timely labyrinth operation. With diffuse suppurative labyrinthitis, advance of the disease can only be recognized by the appearance of symptoms of beginning meningitis. Then the operation is usually too late.

Type.	Previous History.	Symptoms.	Hearing.	Caloric Reaction.	Turning Reaction.	Fistula Symptom.	Indicated Operation.
Circumscribed labyrinthitis.	Vestibular symptoms in attacks.	Vestibular symptoms. Nystagmus to sound side, or to diseased, or to both.	+	+	+	+	Radical mastoid operation.
Diffuse serous secondary labyrinthitis.	Vestibular symptoms in attacks.	Vestibular symptoms. Nystagmus to sound side.	— +	+ —	+ —	+	Radical mastoid operation.
Diffuse serous induced labyrinthitis.	—	Vestibular symptoms. Nystagmus to sound side.	— +	+ —	+ —	—	Radical mastoid operation.
Diffuse suppurative manifest labyrinthitis.	Vestibular symptoms present or absent.	Vestibular symptoms. Nystagmus to sound side.	—	—	—	—	Labyrinth operation.
Diffuse suppurative latent labyrinthitis.	Vestibular symptoms.	—	—	—	— (+ by compensation.)	—	Labyrinth operation (when + by compensation, radical operation only).

FIGURE I.

Circumscribed labyrinthitis. Labyrinthine fistula in the horizontal canal. Bony wall broken. Endosteum intact. Membranous canal intact. Circumscribed inflammation in the perilymph space.

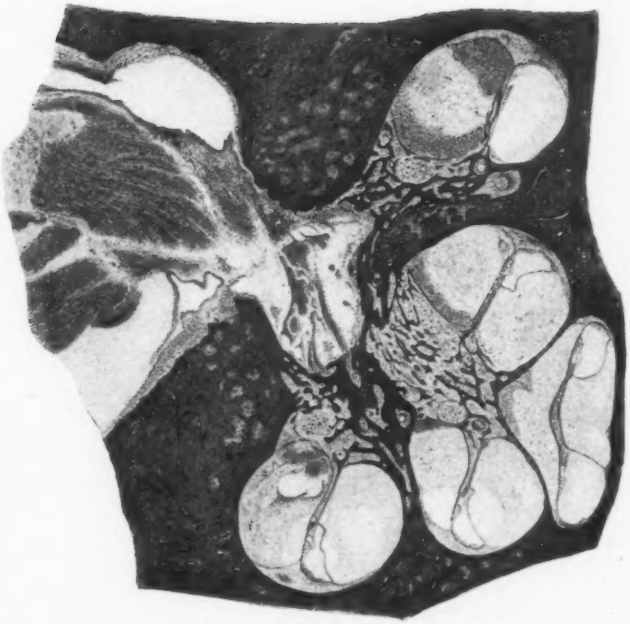

FIGURE II.

Diffuse serous labyrinthitis. Section through the cochlea. Cochlea filled in all turns in the perilymph space by serofibrinous exudate, blood, and only a few leucocytes. Pus in the internal meatus.

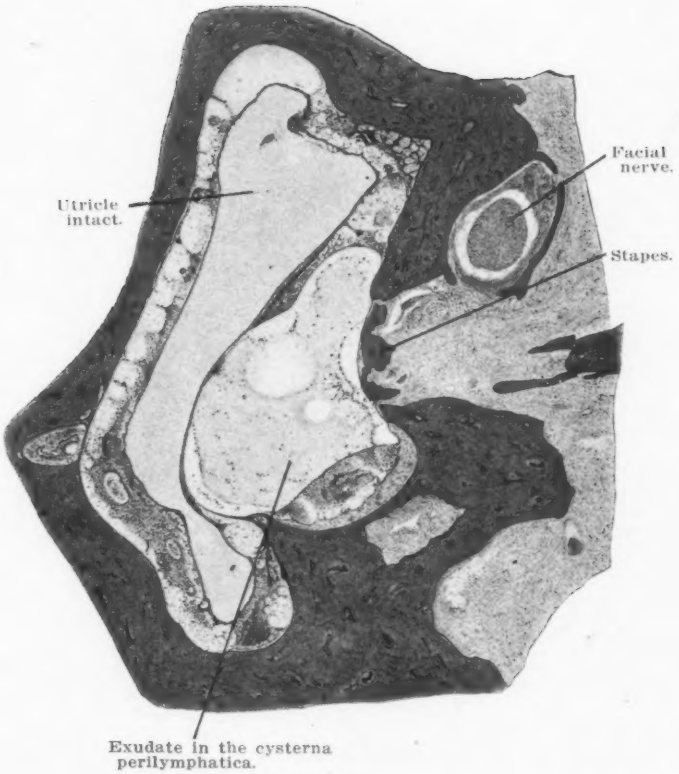

FIGURE III.

Diffuse serous labyrinthitis. Section through the vestibule. (Same case as in Figure II.)

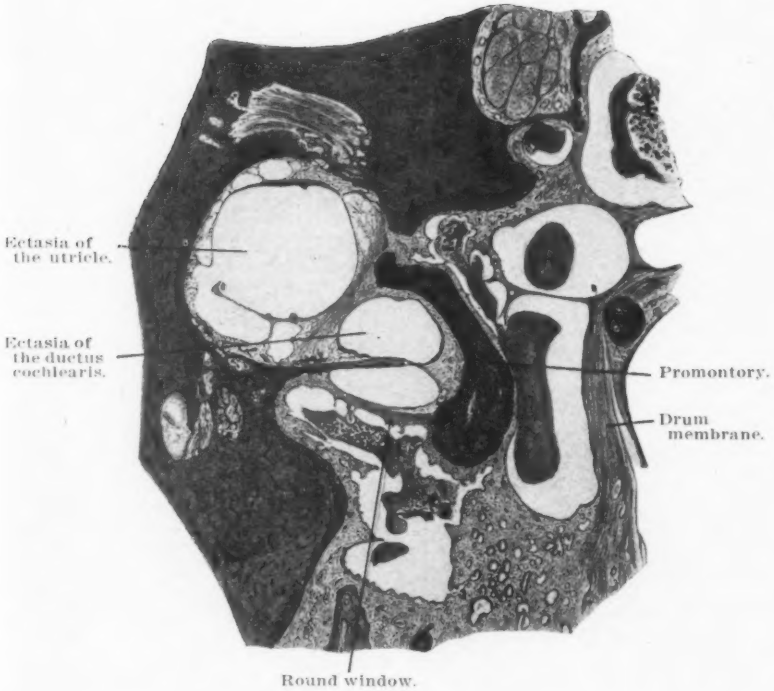

FIGURE IV.

Remains of a serous (?) labyrinthitis. Section through the vestibule.

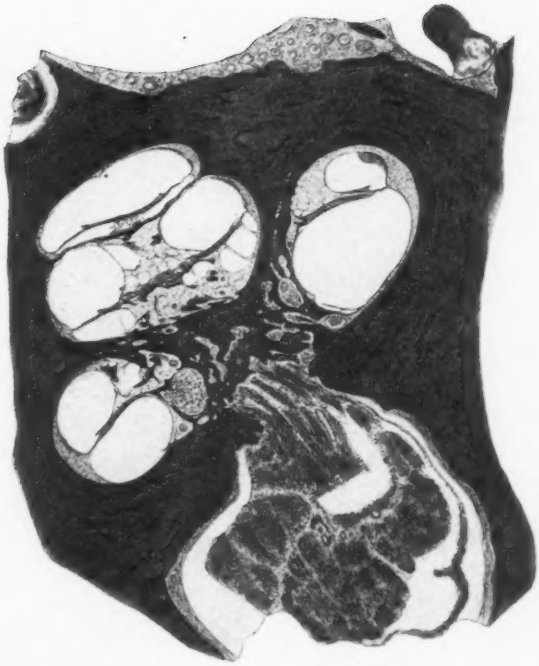

FIGURE V.

Remains of a serous labyrinthitis. Ectasia of the ductus cochlearis. Organized exudate in the scala vestibuli.

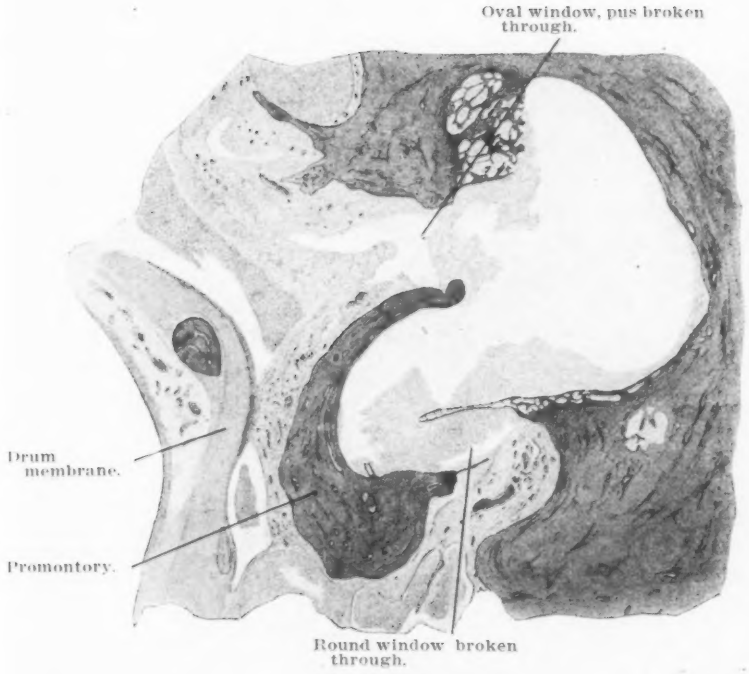

FIGURE VI.

Manifest purulent labyrinthitis.

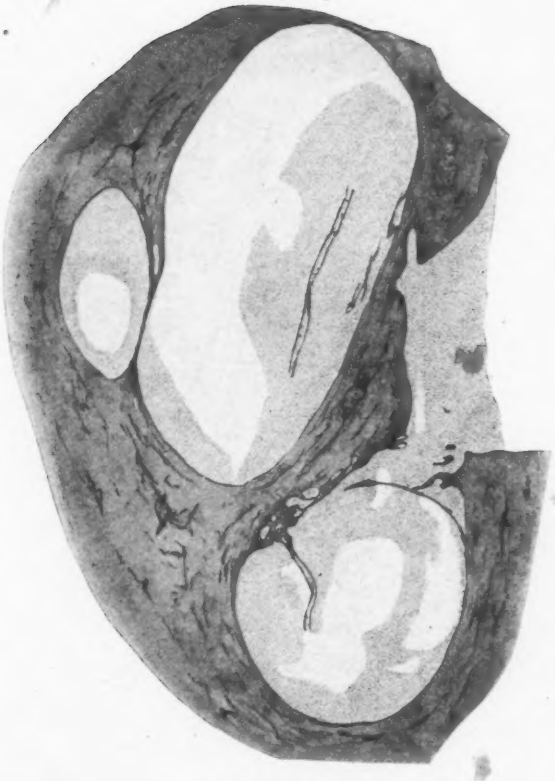

FIGURE VII.

Diffuse purulent labyrinthitis. Section through the cochlea.

XLIII.

A CONSIDERATION OF BRAIN ABSCESS, OF OTITIC ORIGIN, BASED UPON A STUDY OF TWENTY-ONE CASES COMING UNDER THE PERSONAL OBSERVATION OF THE WRITER.*

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A discussion of the subject of abscess of the brain is always of interest to the otologist on account of the comparative frequency of the occurrence of lesions of this character, the obscurity of diagnosis, and the difficulty of bringing the cases to a favorable termination, as the result of operative interference, even when the conditon is recognized.

While the report of the following series of cases may add nothing new to the literature of the subject, these cases represent the work of the author over a period of nearly twenty years, and are therefore presented as a contribution to clinical otology. The individual histories are meager in many of the cases. I have attempted, however, to give an abstract of the history of each case, making this abstract as full as the original notes permit. Important data in many of these histories are wanting, yet, collectively, I believe they are of sufficient scientific importance to warrant their presentation.

Case I.—C. P., adult male. Patient brought to the hospital on May 14, 1898, in a condition of semicoma, with an indefinite history of aural suppuration, involving the right ear. The temperature record has been lost. A large temporosphenoidal abscess was opened by the removal of a large portion of the squamous plate of the temporal bone above the zygoma. The abscess was evacuated through healthy dura. After thorough evacuation of the abscess, the cavity was packed with iodoform gauze. Two days later, as the patient was still in a critical condition, the abscess cavity was again explored by one of my assistants, during my absence from the city, and an abscess

*Read before the Ninth International Otological Congress, Boston, 1912.

of considerable size was found beyond the first abscess. The patient died five days after the operation.

Case II.—A. Z., patient a male child, four years of age, was admitted to the hospital. No history was obtainable, as the child's parents were not seen. The patient was suffering from a mastoiditis upon the right side, and the simple mastoid operation was done. Neither dura nor sinus were exposed at the time of the operation. The patient died four days after the mastoid operation, and a cerebellar abscess was found on autopsy. On examining the mastoid wound, a very small perforation was found in the bone covering the lateral sinus. This perforation led through the sinus, which was thrombosed, into an abscess cavity in the cerebellum. The patient had presented no symptoms of cerebellar abscess aside from the fact that when the dressings were changed, two days after the mastoid operation, the dressing was found to be saturated with foul-smelling pus. This sign alone should have attracted my attention to the fact that there might be a purulent collection within the cranial cavity.

Case III.—D. E., a male, thirty years of age. The patient gave a history of an acute otitis upon the left side, three months prior to admission to the hospital, with mastoid tenderness. Operation upon the mastoid was impossible at this time, on account of a complicating pneumonia. The patient was discharged from the hospital, apparently cured of his mastoiditis, but was readmitted three months after his first admission to the hospital, with a high temperature and in a state of coma. An abscess was found in the deeper portion of the left temporosphenoidal lobe. This abscess was evacuated by the removal of bone above the zygoma, the operation being performed through healthy dura. The temperature fell immediately after the evacuation of the abscess, but there were several sharp rises in temperature during the convalescence, due to small secondary abscesses forming in the brain substance, these abscesses communicating directly with the main abscess cavity.

At the time of the patient's second admission to the hospital, an examination of the fundus oculi showed a choked disc. The patient made a complete recovery. The abscess cavity was at first drained by means of gauze, and later by rubber drainage tubes.

Case IV.—A. P., male, four years of age. The patient gave a history of a left-sided suppurative otitis, of two months' duration. Wilde's incision had been made prior to the patient's admission to the hospital. The left mastoid was opened in the usual manner, neither the sinus nor the dura being exposed. Five days after the mastoid operation, owing to certain indefinite cerebral symptoms, an exploratory operation for cerebellar abscess was performed. The exploration was negative. The patient died of pneumonia thirteen days after admission to the hospital. A temporosphenoidal abscess was found on autopsy.

In this case an error in diagnosis had been made. The patient presented none of the characteristic symptoms of a left temporosphenoidal abscess, but did present symptoms of a suppurative focus within the cranial cavity. In the absence of localizing symptoms, an exploratory operation was performed upon the cerebellum.

Case V.—M. D., male, thirteen years of age, admitted to the hospital in a condition of stupor. The patient was suffering from a suppuration of the left middle ear, the duration of which was unknown. The temperature was between 103° and 104,° and the patient was somewhat aphasic. An ordinary mastoid operation was performed, and at the completion of the operation the dura was exposed above the zygoma, and a large temporosphenoidal abscess evacuated through healthy dura. The abscess cavity was packed with iodoform gauze. The temperature fell to nearly normal, the patient became mentally bright, and the aphasia gradually disappeared. Subsequently, all of the symptoms recurred with increased severity. The brain was further explored, but with negative results. The patient died of purulent meningitis, seventeen days after the first operation.

Case VI.—M. H., female, twenty-nine years of age, mentally feeble, an inmate of an institution for the feeble-minded, and admitted to the hospital suffering from an acute mastoiditis upon the right side. Twelve days after a simple mastoid operation, an opening was found in the tympanic roof, and a probe was passed directly into the middle cranial fossa. Immediate exploration of the middle cranial fossa was performed, and an abscess was found in the right temporosphenoidal lobe. The abscess was evacuated by incision of the dura over the

tympanic roof, the dural exposure having been extended from the tympanic roof to above the level of the zygoma. Smears from the abscess cavity showed streptococci. The abscess cavity was packed with iodoform gauze, and the patient made an uninterrupted recovery. This operation was performed by one of my colleagues, under my personal supervision.

Case VII.—M. F., a young adult, male, twenty-one years old, admitted to the hospital with a history of purulent discharge from the left ear for two years. A radical operation was performed upon the left side, and two weeks later a secondary grafting of the radical cavity was done. There was a rise in temperature the day following the secondary grafting; the posterior wound was opened, the graft removed, and the temperature fell to normal. Subsequently, the temperature rose again, cerebral symptoms developed, the left optic disc became choked, there was some ocular aphasia and homonymous hemianopsia. The left temporosphenoidal lobe was explored and drainage maintained for a number of weeks. The patient continued to improve until about three months after his admission to the hospital, when he suddenly developed symptoms of meningitis, and finally died three and a half months after the first operation.

Case VIII.—I. G., a male, fifty-two years of age, admitted to the hospital with the history of a previous acute otitis media, upon the right side, some weeks before admission. Upon admission to the hospital the patient seemed mentally dull. Both optic discs were normal, and there was some paralysis of the left upper extremity. Examination of the ears showed the right external auditory meatus narrow at the fundus, luster of the drum membrane lost, no bulging of drum membrane, slight indefinite tenderness over the tip of the mastoid. The patient complained of considerable headache. This patient was left handed, and showed the symptom of mirror writing. The mastoid was opened five days after admission to the hospital, and some granulation tissue was found. An abscess located deeply in the right temporosphenoidal lobe was evacuated. The patient's condition improved immediately after operation, the sensorium became clear, and he was able to write. The patient subsequently died of purulent meningitis. In this case the abscess was evacuated through healthy dura.

Case IX.—Mrs. B., about thirty-eight years of age, pre-

sented, having had an acute otitis upon the left side ten years before I saw her. The patient had had a recent attack of acute otitis upon the left side, which had lasted two weeks, before she came under my observation. She presented all the symptoms of an acute mastoiditis, and the left mastoid process was opened forty-eight hours after I first saw the patient. The lateral sinus was exposed during the operation, and the sinus wall, being soft, was opened. There was free hemorrhage. It was noticed that at the beginning of the anesthesia the patient presented some unusual aphasic symptoms. After the mastoid operation the patient made perfectly satisfactory progress for a month, when she suddenly became aphasic, the aphasia being of the "jargon" type. The temperature at the inception of the aphasia was 101.° The patient was kept under careful observation for a period of twelve days, during which period the temperature gradually fell to nearly normal. The patient was seen during this time by several eminent consultants, among whom were a prominent neurologist, and a well-known surgeon, and all agreed that the case was probably one of cerebral thrombosis, and not one of brain abscess. The differential blood count showed 85 per cent polymorphonuclear leucocytes. I advised exploratory operation in this case, but was overruled by the other consultants. Eight days after the beginning of the cerebral involvement the patient had severe headache and an abrupt rise in temperature. Four days later the symptoms became very pronounced, an operation was performed, and about two drams of pus evacuated from the inferior frontal convolution and the island of Reil. A cigarette drain was inserted, but the patient died on the day after the operation. I should state that in this case the pulse had been very slow throughout the entire time that the patient was under observation. There were no changes in the fundus oculi in this case.

Case X.—G. B., a male, six years of age. There was a history of suppuration in the left ear since early infancy in this case. The radical operation was performed, and primary skin grafting employed. The dura was not exposed at the time of the operation. The patient had some temperature immediately after the operation, the wound being septic. These symptoms disappeared, and the patient did well after this until about one month after the operation. The patient had a slow

pulse, however, and there was a slightly irregular temperature elevation, varying between 99.5° and 100.° The patient left the hospital and was treated as an ambulatory case. About one month after the operation the patient had an attack of vomiting, a sharp rise in temperature, and complained of severe headache. He also exhibited considerable mental dullness. In my absence the left temporosphenoidal lobe was explored through the tympanic roof and the adjacent squamous region by one of my assistants. The internal table was found carious over the tympanomastoid roof. The abscess cavity was packed with gauze, and the patient made a complete recovery.

Case XI.—F. P., male, twenty-six eyars of age, presented with a history of chronic suppurative otitis media, involving both ears, since eight years of age. The patient remained under my observation for six years, the otorrhea being kept very well under control. In the spring of 1905 the patient's general health began to be impaired, and he lost flesh steadily, while the suppuration upon the left side seemed to be more profuse than usual. A radical operation was performed upon the left ear in October, 1905, primary grafting being employed. There was no exposure of either sinus or dura. Left facial paralysis appeared three days after the operation, but disappeared almost entirely at the end of a month. The middle ear cavity was dry six weeks after operation, with the exception of a small granulating area in the lower part of the tympanum. A little less than five months after the operation the patient had an attack of nausea and vomiting, double vision, and a recurrence of the facial paralysis. There was marked incoordination of both upper and lower extremities. There was paresis of the left external rectus and of the left superior oblique. The visual field, upon the left side, was contracted, and there was slight congestion of the veins of both optic discs—not, however, sufficient to be called "choked disc." The temperature was subnormal and the pulse varied between 60 and 70. A differential blood count showed a polymorphonuclear percentage of 72 per cent. The pulse gradually fell in frequency, there was some headache and slight rigidity of the neck. The patient gradually became comatose, the temperature rose to 102.8,° the diagnosis of cerebellar abscess was made, and the patient was operated upon. An attempt

was made to explore the cerebellum in front of the lateral sinus, but as the sinus was placed rather far forward, exploration in this region was unsatisfactory, and a more complete exploration was made behind the lateral sinus. The cerebellum was punctured by means of a long knife and explored in various directions by means of a director. No pus was found. The patient's condition immediately after operation was extremely grave, but on the second day the temperature dropped to 99.5,° the patient became conscious, and there were no evidences of any paralyses. On the third day after the operation the temperature gradually rose, the patient became unconscious and died. At autopsy a cerebellar abscess was found at the cerebello-pontine angle. There was also a fibrosarcoma of the left auditory nerve trunk, and the left cochlea was practically entirely destroyed by the neoplasm. A full report of the above case is given in the *Transactions of the American Otological Society*, Vol. X, Pt. 2, p. 266.

Case XII.—B. H., a male, thirteen years of age. The patient when admitted to the hospital was suffering from an acute exacerbation of a chronic suppuration of the right middle ear. There was severe pain about the ear and edema about the mastoid process extending downward into the neck. A radical operation was performed, with evacuation of an epidural abscess over the lateral sinus. The patient did well for two days, when there was slight temperature elevation. A differential blood count showed a polymorphonuclear percentage of 84 per cent and a leucocytosis of 33,600. There was a sinus extending from the lower angle of the mastoid wound downwards into the neck, and this was drained by a counter opening in the neck. The patient progressed favorably for two days more, when his temperature rose to 104,° and he became stupid and complained of severe headache. The cerebellar substance was explored, both in front and behind the lateral sinus, and a cerebellar abscess was found. Through-and-through drainage was established. The patient died twenty-four hours after the operation.

Case XIII.—Miss S. R., age nineteen, was seen in consultation, November 24, 1907. There was a history of chronic purulent otitis media, upon the left side, of ten or fifteen years' duration. The mastoid operation had been done prior to my seeing the patient, and a clot had been evacuated from the lat-

eral sinus. Seven days after the mastoid operation the jugular had been excised. The temperature after excision of the jugular had remained between 100° and 101.° When I first saw the patient the temperature was 99.4° and pulse 80, there were alternating attacks of restlessness and stupor, and continued picking at the face and body. Increased polymorphonuclear count. There was no nystagmus, and the fundus of each eye was normal on ophthalmoscopic examination. The patient had also had considerable headache. Examination of the wound showed a profuse discharge coming from an opening through the dura, just at the knee of the lateral sinus. I was able to pass a probe directly into an abscess cavity in the cerebellar substance. The attending physician, Dr. Dougherty, with my assistance, exposed the cerebellar dura more freely, and freely incised the cerebellar substance, draining a large abscess cavity through the enlarged original opening. At the time of this operation I suggested to Dr. Dougherty that in case this procedure did not relieve all symptoms, a counter opening behind the sinus should be made, and that through-and-through drainage should be established. The patient did well for four or five days, when the headache returned, there was a rise in temperature to 103,° with a rapid pulse. The patient collapsed, became cyanosed and practically pulseless. Normal saline solution was infused, and Dr. Dougherty established through-and-through drainage, as had been suggested by me at the previous operation. The patient made a complete recovery.

Case XIV.—S. R., a male, twenty-nine years of age. There was a history of pain in the left ear, of one month's duration. For three days the left side of the face had been paralyzed. The ordinary signs of acute mastoiditis were present. At operation the antrum was found to be very small, the roof of the antrum was wanting, and the dura in this region was necrotic. A probe could be passed through the perforation in the dura into an abscess cavity in the temporosphenoidal lobe. In order to expose the infected area more thoroughly, a complete radical operation was done, the abscess was opened along the route of infection, and was drained first with a cigarette drain, and later with folded rubber tissue. Four weeks after the primary operation a Thiersch graft was applied to the cavity. The patient made a perfect recovery.

The symptoms in this case are exceedingly indefinite. The patient was an Italian and spoke no English; consequently, it was impossible to determine whether or not there was any aphasia.

Case XV.—J. C., male, twenty-six years of age, was admitted to the hospital with a history that three weeks before admission a simple mastoid operation had been performed upon the left side. The patient had had severe headache for two weeks, the temperature was slightly elevated, the eye grounds were negative, there was no nystagmus, the knee jerk was absent on the left side, and there was some weakness of the left side of the body. The previous mastoid wound was revised. It was found that the antrum had not been opened at the previous operation, but that the dura in the middle cranial fossa had been perforated, and that the examining probe passed directly into the brain substance. The opening in the dura was enlarged, and a cigarette drain inserted into the brain substance. The patient did well for two weeks, and then developed double choked disc. Lumbar puncture was at first negative, but three weeks after the operation lumbar puncture evacuated purulent spinal fluid. The patient, in spite of this unfavorable symptom, continued to improve for two months and a half, when he had a rise of temperature to 101.3.° The patent then ran an irregular temperature for two weeks, and finally died of a purulent meningitis. The brain substance was again explored, but no further collection of pus was found within the brain. At autopsy the right sphenoidal sinus was found to contain pus and the pituitary body was soft and discolored. It is possible that the fatal termination was due to the suppuration in the sphenoidal sinus and not to the middle ear lesion.

Case XVI.—R. H., female, fifty-seven years of age. Five days before admission patient had pain in the left ear; two days later, pain in the right ear. The left ear discharged spontaneously on the third day, and the right on the fourth day. The right mastoid was opened, dura exposed, and a small perforation found in the dura, leading into a cavity about an inch in depth. The left mastoid was operated upon three days later. There was no intracranial involvement upon this side. The patient did badly after the first operation, in spite of drainage of the abscess cavity. The abscess cavity was

again explored, with negative results. The patient died five days after the first operation, with high temperature. The infection, in this case, was streptococcus capsulatus. This germ was found both in the smears from the abscess cavity and in the cerebrospinal fluid.

Case XVII.—M. C., a woman fifty years of age, entered the hospital suffering from an acute otitis upon the right side, severe headache and tenderness over the right mastoid. A complete mastoid operation was done, the sinus and dura both exposed at the time of the operation. The sinus was located very far forward. There was very little destruction found in the mastoid, and for this reason the wound was completely closed by sutures. Three weeks after the operation the patient had a rise in temperature, and there was considerable mental dullness. Ophthalmoscopic examination showed slight congestion of the optic discs. The polymorphonuclear percentage was 68.6 per cent. Owing to the cerebral symptoms and the rise in temperature, a decompression operation was performed over the right temporosphenoidal lobe, and an abscess evacuated. The patient died on the day after the operation. In this case the drainage was secured through healthy dura, no direct avenue of infection being demonstrable.

Case XVIII.—M. L., a woman twenty-seven years of age, suffering from chronic suppurative otitis media, upon the right side, was operated upon by one of my assistants. The radical operation was performed, the dura in the middle cranial fossa being exposed at the time of operation. No skin grafting was employed. The patient ran an irregular temperature after the operation, and complained of some headache. Two and a half weeks after the operation the patient became suddenly comatose. Ophthalmoscopic examination showed the fundus of each eye to be normal. Lumbar puncture evacuated a clear, sterile cerebrospinal fluid. The differential blood count showed 86.4 per cent of polymorphonuclear leucocytes. I was inclined to believe that the case was one of serous meningitis, and removed a large portion of the squamous plate, with the idea of doing a decompression operation. The removal of bone was made to include the roof of the external auditory meatus, in order to make the decompressed area continuous with the area of dura exposed at the time of the original opera-

tion. Examination of the dura showed a mass of granulation tissue covering the dura, over the area that had been exposed at the primary operation. A director passed easily through this granulation tissue into the substance of the temporosphenoidal lobe, and the entrance of the director evacuated a large amount of foul-smelling pus. The opening in the dura was enlarged, and the brain abscess cavity completely drained. The patient died on the following day. In this case the abscess was drained along the avenue of infection. Unfortunately, the symptoms had been so obscure as not to attract much attention, otherwise, I believe this patient might have been saved, as the abscess was easily accessible, and was located in an ideal position to secure drainage.

Case XIX.—A. F., a male, seven and one-half years old, operated on November 30, 1910, for simple, right-sided mastoiditis, by one of my assistants. Two days later the pulse suddenly became slow, ranging between 40 and 50, the patient had a convulsion and became comatose. A rapid exploratory operation was done by one of my assistants, during my absence, a large decompression operation being performed, and the brain substance explored with negative results. Complete recovery followed the decompression operation. On September 2, 1911, the same patient was brought to the hospital, unconscious, there was congestion of the optic discs, and the spinal fluid was negative. A differential blood count showed a polymorphonuclear percentage of 89 per cent. The old decompression wound was reopened by one of my assistants, and an abscess of considerable size was found in the temporosphenoidal lobe. The abscess cavity was drained, and the patient made a complete recovery.

Case XX.—H. M., a girl of sixteen, was admitted to the hospital with a history of discharge from the left ear, of several years' duration. For some days the patient had suffered from severe headache in the left side of the head, and tenderness over the mastoid. The temperature was about 101,° and the patient seemed very ill. A radical operation was performed, the sinus exposed, and an area of deeply congested dura was also exposed in the middle cranial fossa. A plastic meatal flap was formed, and the posterior wound sutured, no graft being applied. The patient vomited considerably for two days after the operation. The vomiting was thought to be due to the

anesthetic. The temperature remained between 100½° and 101½°. The patient also complained of severe headache, and seemed very ill. Four days after the operation the temperature was 101,° the patient was dull and slightly delirious. The pulse at this time was 48. An examination of the chart showed that the pulse rate had gradually been falling, and that this retardation had become rapidly more pronounced within the last few hours. An ophthalmoscopic examination showed beginning choked disc upon the left side. The polymorphonuclear count was not above 80 per cent. The patient presented the characteristic optic aphasia, being unable to name objects presented to her, although she was able to designate the use of such objects. The original mastoid wound was reopened and enlarged, a large portion of the squamous plate was removed, and the dural area exposed was made continuous with the dural area exposed at the first operation. The dura was incised over the thickened area exposed at the primary operation, and exploration of the brain substance, by means of the director, revealed an abscess cavity of considerable size in the temporosphenoidal lobe. About three ounces of extremely foul pus was evacuated. A cigarette drain was introduced into the cavity. Later a rubber tissue drain was substituted for the cigarette drain. The patient made a complete recovery. The infection in this case was pneumococcus.

Case XXI.—A. P., female, colored, thirty-eight years of age. There was a history of syphilis in this case, and syphilitic caries of the hard palate. There was an extensive subperiosteal mastoid abscess upon the left side, apparently of acute origin, and left facial paralysis. An extensive mastoid operation was done, with the exposure of a large dural area in the middle cranial fossa. The zygomatic cells were very extensively involved. A portion of the posterior canal wall was taken away, and the ossicles were removed, but the complete radical operation was not done. The patient was immediately put upon hypodermic injections of mercury. She improved after the operation, but an area of bare bone remained corresponding to the floor of the middle cranial fossa anteriorly, and the dura was thickened. One month after the operation, on dressing the wound, the dura of the middle cranial fossa was found to be completely necrotic over a considerable area, and the probe entered the brain substance. A

cavity as large as a pigeon's egg was found in the temporosphenoidal lobe. This cavity was drained by means of a cigarette drain and the radical operation completed. The patient made a complete recovery. This patient had presented absolutely no symptoms of temporosphenoidal abscess, aside from a slight elevation of temperature to 101,° about two days prior to the discovery of the abscess. She had been repeatedly examined for aphasic symptoms, but they were absent. The pulse had been normal in frequency, and the patient complained of no headache. The field of vision had been slightly contracted when the patient came to the hospital, which was thought to be due to her specific infection. An ophthalmoscopic examination revealed congestion of both of the discs with an abnormal tortuosity of the veins upon the left side.

From the histories of the collated twenty-one cases, it will be noticed that temporosphenoidal abscess occurred much more frequently than did cerebellar abscess. There were seventeen cases of temporosphenoidal abscess, while cerebellar abscess was found in only four instances. From this series of cases it would appear that infection of the temporosphenoidal lobe is four times as frequent as infection of the cerebellum. My experience here is the reverse of that of some writers, who have found that cerebellar abscess occurs about twice as frequently as does temporosphenoidal abscess.

Regarding the side affected, out of seventeen cases of temporosphenoidal abscess, seven occurred upon the right side, and ten upon the left side. Of the cerebellar abscesses, two cases occurred upon the right side and two upon the left side.

The avenue of infection in the cases of temporosphenoidal abscess seemed to be fairly well marked in a number of instances. In nine cases infection occurred through the tympanomastoid roof. One case, in which the abscess was found in the island of Reil, was evidently of metastatic origin. In another instance the brain abscess occurred several months after a decompression operation for the relief of cerebral symptoms, and at the time of the decompression operation the temporosphenoidal lobe was explored for a possible abscess. As the purulent discharge from the ear still continued, it seems probable that infection occurred in the brain substance, which had been partially broken down following the preceding exploration. The abscess, in this case, was superficial, and

occupied the area previously explored. In one of the cases, where the tympanoantral roof was the avenue of infection, it is possible that the abscess was the result of the breaking down of a gummatous deposit. In three of the cases of cerebellar abscess infection occurred through the lateral sinus. In the fourth case the abscess was complicated by a neoplasm of the auditory nerve trunk. The cochlea was entirely destroyed, and it is probable that infection occurred through the internal auditory meatus, although this could not be clearly demonstrated, owing to the invasion of the internal ear by the neoplasm.

Regarding the frequency with which these cases followed acute and chronic middle ear suppuration, respectively, the cases reported are of interest. In four cases the duration of the suppuration was unknown; in seven there was a history of chronic suppuration, while in ten intracranial involvement seemed to follow an acute suppuration of the middle ear. In two of the cases following acute midde ear suppuration there was a period of latency—in one case apparently extending over ten years, the abscess being excited to renewed activity by a second attack of otitis media. In the second case the period of latency was about one month.

These cases, therefore, seem to give a relatively high percentage of brain abscess following acute middle ear suppuration. In four cases following acute otitis, the antrum was exceedingly small and the middle fossa very low. This anatomic fact may explain the reason for the development of a suppurative focus within the temporosphenoidal lobe following an acute otitis, in these particular cases. In one of the cases, in which a mastoid operation had been done before the case came under my observation, and in which the antrum was very small, the operator had entered the temporosphenoidal lobe under the impression that he had opened the antrum. Such an error is, of course, not unheard of, and the occurrence of this accident is mentioned to emphasize the importance of the operator satisfying himself beyond any doubt, in every case of acute mastoiditis, that he has really entered the mastoid antrum, and has not perforated the tympanic roof and invaded the midle cranial fossa.

The symptomatology presented in the series of cases is worthy of analysis. Some of the patients were brought to

the hospital in a semicomatose condition, and, consequently, a detailed record of symptoms is not given in the histories.

The most constant symptom, I should say, was headache. This was noted in most of the cases in which the patients were not comatose at the time of admission, with two exceptions. It was absent in one case of temporosphenoidal abscess of specific origin, and also in the case of cerebellar abscess complicated by a sarcoma of the auditory nerve trunk.

Vomiting occurred in some of the cases, but in many it was not a prominent symptom until the late stage.

The mental condition of the patients varied considerably. In four of the patients coma was the most prominent mental symptom. Nine of the patients were exceedingly dull mentally, although they could be easily roused from their condition of semistupor, and when so roused answered questions fairly intelligently. The sensorium was normal in two cases. In one case the patient was very neurotic, and in a state of extreme nervous excitability. Three of the patients were lacrimose, their condition bordering on one of melancholia. They wept when discussing their physical condition, and seemed to feel certain of a fatal outcome. I mention this symptom particularly, because in all of these three cases the condition was of long duration, the patients were under observation for a considerable period of time, and the state of mental depression was extremely characteristic. In all of these cases the abscess was located in the left side of the cerebrum, two being cases of abscess of the temporosphenoidal lobe, while in the third case the abscess was situated in the island of Reil. In one of the patients in whom the sensorium is accounted "normal," the patient came from an institution for the feeble-minded, so that any change in the mentality of this patient was difficult of observation. In two of the cases the mental condition is not noted.

Muscular paralyses did not occur very frequently in these cases. So far as I have been able to determine from my notes, muscular paralyses were present in only two cases, namely, in one case of abscess of the right temporosphenoidal lobe, in which there was some paralysis of the left upper extremity (Case VIII), and in another case (Case XI), one of cerebellar abscess, in which there was paralysis of the external rectus, upon the affected side.

Convulsive seizures were also rare. A general convulsion was noted in the early history of Case XIX, the convulsive seizure involving the opposite side of the body. This occurred, however, before the abscess was discovered, although it may have been present at that time.

The condition of the fundus oculi is also interesting. Out of the twenty-one cases, in six the condition of the optic disc is not given; in three cases there was choked disc, in three congestion of the optic disc, and in the remaining cases the ophthalmoscopic examination was negative.

Regarding the occurrence of aphasia, three cases of temporosphenoidal abscess showed this symptom. In these cases, naturally, the abscess occurred upon the left side; five cases of left-sided temporosphenoidal abscess showed no aphasia; two of these five cases were Italians, who spoke practically no English, and, consequently, the determination of this symptom would have been rather difficult. Moreover, the abscess in each of these cases was found at the time of operation, and no careful test had been made for the symptom prior to the operative interference. In one of the cases suffering from a temporosphenoidal abscess upon the left side, the patient was comatose when admitted to the hospital, and it was impossible, therefore, to determine the presence or absence of this symptom. This patient was a Hebrew, who spoke no English, and the determination of aphasia during convalescence was consequently impossible. In one case of left temporosphenoidal abscess, the presence or absence of the symptom of aphasia is unnoted. In the remaining seven cases of temporosphenoidal abscess, the lesion occurred upon the right side, and, therefore, no aphasic symptoms would be expected. One of these patients, however, was left-handed, and did present the characteristic symptom of agraphia, the agraphia, in this particular instance, taking the form of mirror-writing. In the case where the abscess was located in the inferior frontal convolution, and in the island of Reil, the aphasia was of the "jargon" type, characteristic of a lesion in this location.

The temperature record in these cases is rather interesting. In two of the cases the temperature record has been lost; in four cases the temperature was high, that is, it was about 104° when the cases presented themselves for operation. In the

other cases there was a moderate temperature, that is, a temperature varying between 100° and 102°. In one case, that of specific origin, there was no temperature elevation, excepting one rise to 100° F. In one other case the temperature was high at the time of operation, and moderate during the period of latency.

This review of the temperature record, presented by these cases, is of considerable interest, in that, given a case of middle ear suppuration, with headache and a slightly irregular temperature, that is, a temperature varying between 99° and 102,° the possibility of cerebral abscess should always be borne in mind. The presence of an epidural collection of pus could naturally easily be excluded, either at the time of the original mastoid operation or by subsequent exploration; by the removal of more bone about the area of already exposed dura. When this is done, and the symptoms of headache and moderate temperature continue, the operator should be continually on the alert for further evidences of infection of the brain substance.

Concerning the pulse rate, in six cases there is no record of the pulse rate noted. In six cases the pulse was slow, and in nine cases the pulse showed no characteristic variation, the pulse-temperature ratio being about normal.

A differential blood count was not made in many of the earlier cases. In some of the later cases, as will be seen by a perusal of the histories, the brain abscess was discovered on the operating table, and no differential blood count was made previous to a discovery of the condition. In eight cases where a differential blood count was made, in three cases the polymorphonuclear percentage was found to be above 80 per cent, while in five cases the polymorphonuclear percentage was not increased above the normal standard. It would seem, therefore, that a cytologic examination is of little value in making a differential diagnosis in cases of this character. My opinion is, that where, in a suspected case, the polymorphonuclear percentage is increased, this sign should furnish strong confirmatory evidence of the presence of a brain abscess, if this condition is suspected. On the other hand, the findings in this series of cases seem to show that the absence of an increased polymorphonuclear percentage in no way militates against the diagnosis of brain abscess.

Regarding the value of lumbar puncture, very little can be learned from the series of cases reported. Lumbar puncture was not employed in the earlier cases, owing to the fact that it was not at that time recognized as a measure of diagnostic importance. Out of the twenty-one cases reported, lumbar puncture was employed in four cases. In two of these cases the cerebrospinal fluid was sterile. In one case the fluid obtained by lumbar puncture showed streptococcus, and in the second case, streptococcus capsulatus. Both cases showing infectious organisms in the cerebrospinal fluid terminated fatally. A negative finding in the cerebrospinal fluid, however, does not necessarily mean that the prognosis is good, as one of the cases in which the cerebrospinal fluid was negative, and in which a large temporosphenoidal abscess was evacuated, terminated fatally within twenty-four hours after the operation (Case XVIII). In the second case, where the cerebrospinal fluid was negative (Case XIX), the patient recovered.

We might say, then, of lumbar puncture, that we should expect to find the cerebrospinal fluid under pressure in all cases of brain abscess where the abscess has attained a considerable size. If the cerebrospinal fluid contains pathogenic organisms, this would seem to indicate a rupture of the abscess into the ventricles, or, at least, a diffusion of the infection into the arachnoid space, and, consequently, would render the prognosis grave. The fact, however, that the cerebrospinal fluid is sterile, should not influence one to give an absolutely favorable prognosis, as in one of the cases where the cerebrospinal fluid was sterile, the patient subsequently died.

A correct diagnosis, in these cases, was not especially difficult. In only three cases was the diagnosis delayed. In Case II the condition was found on autopsy. The only symptom of cerebellar abscess which this patient presented was the presence of a considerable amount of foul-smelling pus in the first dressing, following the mastoid operation. In every case in which a complete mastoid operation is performed, and where the disease is limited to the mastoid, no pus should be found upon the dressings when these are changed for the first time. A large amount of purulent secretion in the dressings indicates that the operation has been imperfectly performed. I remember distinctly calling the attention of my

assistant to the large amount of pus in this case, and was at a loss to account both for the quantity of the pus and for its foul odor. At the autopsy a small opening was found in the vitreous plate covering the lateral sinus, and this opening led through the sinus into the cavity of the cerebellar abscess. As this was one of the earlier cases which came under my observation, perhaps I may be pardoned for having overlooked a sign which at the p resent time would not have escaped my observation. In Case IV also—a case occurring early in my experience—a diagnosis of intracranial suppuration was made, and an exploratory operation for cerebellar abscess was performed five days after the mastoid operation. This patient died of pneumonia, and on autopsy a temporosphenoidal abscess was found. In this case also, the diagnosis was at fault. In Case IX, a tentative diagnosis of abscess of the brain was made on the symptom of sudden aphasia. This patient was seen by several prominent general practitioners, one neurologist of international reputation, and by a general surgeon. I was inclined to believe the case to be one of abscess of the brain, but all of my confrères decided against the advisability of operation, and considered the case to be one of cerebral thrombosis. This diagnosis was adhered to until the day before the death of the patient, when symptoms of general meningitis made their appearance. Immediate operation was performed, and an abscess in the inferior frontal convolution and the island of Reil was successfully evacuated. The patient died, however, from meningitis, owing to the fact that the operation had been too long postponed. Here the diagnosis was correct, but operative treatment was not instituted promptly enough to be of avail. In Case XI, the diagnosis was correct, that is, the diagnosis of cerebellar abscess was made. The abscess was not found at the time of operation because the exploring director was not carried deeply enough into the cerebellar substance. The specimen obtained upon autopsy revealed the fact that had the instrument been carried an eighth of an inch further, this cavity would have been opened. Here, the fault was one of operative technic and not of diagnosis. In Case XVIII, the diagnosis was questionable, in that it lay between cerebral abscess and serous meningitis. In this case the ophthalmoscope showed no choked disc upon either side, there was a polymorphonu-

clear count of 86.4 per cent, and lumbar puncture evacuated a clear, sterile fluid. The patient had run an irregular temperature after operation, and became comatose quite suddenly. I was inclined to believe the case to be one of serous meningitis, and exposed the dura over the squamous region and tympanic roof, preparatory to doing a decompression operation. Upon examining an area of dura over the tympanomastoid roof, which had been exposed at the time of the primary operation, I found a mass of granulation tissue present, and on probing this area with a director, the instrument passed readily into the temporosphenoidal lobe, and evacuated a large amount of pus. In this case a correct diagnosis was made upon the operating table, and, consequently, it cannot be classed as one in which any actual error in diagnosis occurred.

Of the cases cited, therefore, an incorrect diagnosis or a failure to make a diagnosis, occurred in only two cases out of the twenty-one, namely, in Case II and in Case IV.

The presence of an abscess, therefore, within the brain substance can be recognized with a considerable degree of certainty. The two cases in which an incorrect diagnosis was made occurred in the earlier years of my practice, before as much was known about brain abscess as is known at the present day. I hardly think that either of the above cases would escape the otologist of today.

The result of treatment, in these cases, is interesting.

Of the temporosphenoidal cases, ten died and seven were cured. Of the cerebellar cases, three died and one recovered. The mortality, therefore, in the cases of temporosphenoidal abscess, was about 58 per cent, while the mortality in the cases of cerebellar abscess was 75 per cent The number of cases of cerebellar abscess reported is comparatively small, but I believe that the mortality is much higher where the abscess is located in the cerebellum than where it is located in one of the temporosphenoidal lobes.

Comparing the above results with a series of cases previously collected, and published in the *Transactions of the American Otological Society* for 1907, I find that out of 102 cases of cerebellar abscess collated from literature, 67.6 per cent of the cases died. As men are more prone to report their successful cases than those which terminate fatally, perhaps the mortality in this small series of mine is not unduly high.

Case IV died of pneumonia, and Case XI was complicated by a neoplasm of the auditory nerve trunk. All of the other fatal cases, in this series, died of a purulent meningitis.

We next come to the proper operative technic to be employed in cases of temporosphenoidal and cerebellar abscess. Of the temporosphenoidal abscesses, one was found on autopsy, as before stated, an exploratory operation having been performed for cerebellar abscess, owing to an error in diagnosis. Of the sixteen cases remaining, in eight the abscess was opened above the zygomatic process, through healthy dura, and of this number, six died and two recovered. In the other eight cases, the abscess was evacuated through the tympanic roof, that is, along the avenue of infection, and of these, five recovered, and three died. Of the cerebellar cases, one was found at autopsy, and two were drained both in front and behind the lateral sinus, and of these two, one recovered and the other died. In the fourth case an attempt was made to evacuate the abscess behind the lateral sinus. The abscess cavity was not entered, owing to the fact of its great depth. The autopsy revealed the fact that had the exploring instrument been introduced an eighth of an inch deeper, the abscess cavity would have been entered.

Regarding the operative technic, therefore, it would seem that the best results would be obtained in any brain abscess by opening the abscess along the path of infection. In this way the arachnoid space is not opened, as the arachnoid space is obliterated for some distance about the path of infection. The histories of the preceding cases show clearly that in cases where the abscess was successfully evacuated, a number of the patients died from purulent meningitis, owing undoubtedly to an infection of the arachnoid space after operation. If the possibility of this infection can be avoided by opening the abscess along the avenue of infection through the amalgamated brain coverings, the chances of recovery will be much greater. This idea has been particularly emphasized by Mr. Ballance, and the cases in this series seem to bear out the wisdom of this plan of procedure; that is, eight cases were opened through healthy dura, and six died. Of eight cases opened through the tympanic roof, that is, along the avenue of infection, only three died. It is only fair to say that of the eight cases opened through healthy dura, in

most of the fatal cases the abscess was much larger than in the second series of eight cases, opened through the tympanic roof. The difference in mortality, however, is so striking that it is worthy of attention. Unfortunately, it is not always possible to discover the avenue of infection in these cases. The patient may have all of the symptoms of brain abscess and certain localizing symptoms may be present, yet careful investigation of the tympanic and antral roof and of the bone covering the sinus and the cerebellum, may show no avenue through which the infection has evidently occurred. Removal of overlying healthy bone may show no dural discoloration. In such a case, the surgeon must at least explore through healthy dura. In instances of this kind I believe that the best results will follow a decompression operation in the middle cranial fossa in cases of suspected abscess above the tentorium, and a similar decompression operation behind the lateral sinus in cases where there are evidences of an abscess below the tentorium. After the dura has been exposed over a considerable area, either in the middle cranial or cerebellar fossa, the dura should be divided by two incisions crossing each other at right angles. Iodoform gauze should then be packed between the dura and pia, so as to thoroughly obliterate the arachnoid space. The effect of this decompression operation and the subsequent packing is twofold: the presence of the gauze will effectually wall off the arachnoid space in the course of from twelve to twenty-four hours. The decompression will favor the progress of a deep-seated purulent collection toward the cerebral surface over the decompressed area—that is, the pus will travel along the line of the least resistance. In from twelve to twenty-four hours after the decompression operation, the brain substance can then be explored over the area of decompression, and if an abscess is found, its evacuation will not cause an infection of the arachnoid space, as this space will have already been fairly well walled off.

In cases of cerebellar abscess certain symptoms pointing to the labyrinth as the avenue of infection may be present, and in these cases the complete radical operation, with exposure of the dura and incision of the cerebellar coverings in front of the lateral sinus, will probably be a method of procedure preferable to a similar operation posterior to the lateral

sinus. The space in front of the lateral sinus is so limited, however, that efficient decompression in this region is difficult to obtain; for this reason it may be desirable in certain cerebellar cases, even where the radical operation is necessary, to perform a decompression operation, both in front and behind the sinus.

Unfortunately, I have no cases which will demonstrate the efficiency of this proposed plan of procedure. I shall certainly attempt this plan, however, in every case of brain abscess which comes under observation, in which the avenue of infection cannot be made out.

Granting that the focus of pus in the brain has been discovered, the exact manner in which the abscess shall be drained is a matter of importance. I believe that I lost a number of cases earlier in my experience on account of too much meddling with the abscess. We must remember that in every case of brain abscess two factors are present which may determine a fatal sequence of the disease. One of these is septic infection of the brain, and the other, and most important, is increased intracranial pressure, due to the presence of the purulent collection within the cranial cavity. Against the first of these factors nature is not slow to throw out barriers; in other words, as soon as a purulent focus occurs in the brain substance, the natural process is the formation of a limiting wall to this purulent collection. If the surgeon, therefore, simply opens the abscess and allows the pus to escape, he relieves the second factor which may lead to a fatal termination, that is, increased intracranial pressure, and he may well leave the other factor, that is, the extension of the abscess to adjacent portions of the brain, to the natural reparative powers of the patient. I am quite certain that in some of my earlier cases the patient died because I was too anxious to thoroughly explore the abscess cavity, and to clean out every little space which might be the seat of suppuration. We must remember that the suppuration has gone on in the brain substance for some time before its discovery by the surgeon, consequently the limiting wall has been more or less completely thrown out, and if the abscess is drained externally, and free drainage is maintained, there is no reason why the abscess should extend more deeply into the brain substance. For this reason it has been my practice in my

later cases to proceed as follows: The brain substance is explored over the suspected area by means of a grooved director, thrust into the brain substance in varying directions. As soon as the director enters the abscess cavity, and the operator becomes cognizant of this fact by the flow of pus along the groove of the director, a pair of dressing forceps is carried along the director until the abscess cavity is entered; the blades of the forceps are then separated. This allows the escape of pus. The forceps is then removed, and a thin, flat retractor is introduced along the director until the end of the director in the abscess cavity is reached. This retractor is then entrusted to an assistant, the director is removed, and a second retractor, an exact counterpart of the first, is passed along the blade of the first retractor until it also enters the abscess cavity. By separating the blades of these retractors, the pus from the abscess is gradually evacuated. It is important not to allow the pus to flow too rapidly, as this may cause such a sudden diminution of intracranial pressure as to favor a hemorrhage into the brain substance. If the pus pours out too freely, therefore, traction on the retractors should be diminished, in order to prevent the too sudden evacuation of the pus. After the abscess has been completely evacuated, gentle separation of the retractors will permit of an inspection of the abscess cavity. This may be carefully wiped out with pledgets of cotton, extreme care being taken not to injure the wall of the abscess cavity. Where the abscess is large I am in favor of introducing the finger carefully into the abscess cavity. If the abscess is small, however, this procedure should not be employed.

Next, we come to the proper drainage. In some of my earlier cases the cavity was packed with gauze. This, I believe, is a mistake, excepting in cases where the abscess is a chronic one and has very unyielding walls. One case which terminated successfully was drained by means of rubber drainage tubes, although I have not employed these for a number of years. At the present time, upon the evacuation of a brain abscess, I pass a cigarette drain, varying in diameter according to the size of the abscess, to the bottom of the cavity. This drain remains in position from twenty-four to forty-eight hours, its removal at the end of twenty-four hours depending upon the condition of the patient. If the tem-

perature is down and the patient's condition improved, the cigarette drain is not disturbed for forty-eight hours. At the end of this time it is removed, the lips of the wound in the brain substance separated by means of retractors, the interior of the abscess cavity inspected and wiped out with pledgets of sterile cotton. Ordinarily, upon the removal of the cigarette drain, considerable pus will escape. The cigarette drain is replaced after the abscess cavity is cleansed, and the dressing changed again in twenty-four or forty-eight hours. In the course of from five to ten days the brain substance will collapse into the abscess cavity, so as to completely obliterate this, and no pus will follow the removal of the drain. As the cavity grows smaller the cigarette drain is replaced by a drain of folded rubber tissue, and this is gradually reduced in length until the entire abscess cavity has obliterated itself. This plan of drainage has been more successful at my hands than has any other, and will, I believe, enable us to save many cases which might be lost if they were too vigorously interfered with in the hope of cleansing every remote crevice of the cavity.

I regret exceedingly that the histories of my cases have not been more complete, that my results have not been better, and that I cannot lay down more exact rules for the treatment of these cases. The preceding remarks are the result entirely of personal experience, and the deductions drawn are the result of a careful consideration of my own cases.

XLIV.

THE EUSTACHIAN TUBE IN CHRONIC OTITIS MEDIA.

By E. M. Holmes, M. D.,

Boston.

In presenting this subject to a society of otologists, it is not necessary to dwell upon the frequency of the chronic middle ear inflammations, nor the resulting affliction to the individuals attacked. The loss of hearing and the suffering from tinnitus is practically ruining the lives of thousands, and anything which we can do to alleviate these distressing symptoms is of the utmost importance. As in a large percentage of the cases the onset is incipient and the progress slow, many have advanced for months and even years before the symptoms become distressing, and often when the otologist is first consulted there are many permanent as well as progressive changes found. Many patients lose from three-fifths to four-fifths of their hearing before they realize or heed their condition. It is, therefore, one of our duties as otologists to impress upon the general practitioner—the family guardian against disease—the importance of testing the hearing of all those who come under his care, whenever possible. The testing of the hearing of all public school children in our cities is doing much to early detect faulty hearing, and we are enabled to treat many during the earlier stages and not only to relieve the existing condition but to diagnose and remove etiologic factors which, if left to progress, would in later years produce distressing results.

Toynbee's work, though half a century old, remains today as a classic in otologic pathology. Lucae, Politzer, Siebenman and others have verified and added somewhat to Toynbee's work. At the present time most otologists divide the nonsuppurative diseases of the middle ear into two classes: the simple hypertrophic, and the sclerotic. It is also generally

conceded that the hypertrophic may become the sclerotic and that the two may coexist. Lucae maintains that in the great majority of cases sclerosis proceeds from a previously existing suppurative or catarrhal middle ear disease. Politzer lays considerable stress upon a secretory stage, and now that we can observe the eustachian tube after opening its lumen, I think we are more impressed with Politzer's statement; for many cases which show no signs of secretion through the membrana tympani, show secretion coming from the tube after application of cocain and adrenalin to its lumen. As it is impossible, with our present knowledge, to decide to what degree the loss of function and the distressing symptoms are due to an existing hypertrophy or to a coexisting sclerosis, it is more certainly impossible to give with any degree of certainty a positive prognosis. It seems very improbable that a true hyperplasia of bone about the footplate of the stapes or in any of the articulations within the tympanum will be absorbed, and yet we have to admit that there are many cases diagnosed by good clinicians as otosclerosis which improve under treatment; and there are other cases which are apparently due to a diseased epipharynx and eustachian tube, which are not relieved by restoring the tube to its physiologic functions. I emphasize this to show that we still lack methods of finding clinically the minute pathologic changes within the ear, and that we have not performed our whole duty to any patient until we have so far as possible removed the causes for a middle ear catarrh, though there may be signs which suggest the more serious condition—otosclerosis. Three of the cases I am reporting in this series had been diagnosed as otosclerosis after they had progressed unfavorably under a long course of treatment, but they were relieved by treatment of the eustachian tube and were, therefore, of hypertrophic and not of sclerotic type.

Since the beginning of modern otology it has been realized in a degree that pathologic conditions within the ear were often due to disease within the eustachian tube, and during the last thirty years the pathology of the nose and its effect upon the hearing has received the careful study of a large number of rhinootologists. Many of these investigators seem to have given too much importance to the nasal pathology as the etiologic factor in middle ear inflammations, for there

are many cases of severe aural disease in which the nose shows no pathologic change, but in many of these we find some trouble in and about the tube. As a result of rhinologic study, marked advance has been made in the treatment of the existing aural disease. Considerable study has also been applied to the epipharynx, and Dr. Wm. Meyer, in calling the attention of the medical profession to adenoid hypertrophies, undoubtedly accomplished more to prevent and relieve middle ear catarrh than any other investigator. After eliminating the large number of cases due to adenoid obstruction which have been relieved by operation, there have been many which have not shown signs of adenoid hypertrophy, and many which have progressed after the usual adenoid operations have been performed. After all the work upon the nose and throat we find that at the present time a large percentage of our aural patients seek relief from the symptoms of chronic middle ear catarrh. Not until the last two or three years has any extensive clinical study been given to the eustachian tube, and we all know that this tube is the tunnel through which the middle ear obtains the air to replace that which is constantly absorbed into the circulating blood, and through which any excessive middle ear secretion drains into the throat. This tube is responsible for the great majority of all middle ear inflammations, and its pathology increases the middle ear conditions in nearly all of the cases due to general systemic causes. It is the main purpose of this paper to present in a general way the pathologic conditions found in the eustachian tube in cases of chronic middle ear inflammation and to give the results upon the aural symptoms by treatment of the eustachian tube.

A spur or deflected septum, a hypertrophic or atrophic turbinate, a purulent or nonpurulent inflamation of the Schneiderian membrane, does not always produce like conditions or of the same degree within the middle ear. Although this is not so marked in pathologic conditions of the eustachian tube, it is true that these conditions in the tube may be associated with slight changes in the middle ear, and comparatively slight conditions within the tube may produce severe processes in the ear. There are a number of obvious reasons for this. Hypertrophy or simple swelling of a large patent tube will not close the lumen to as great a degree as in one anatomically narrow. Nor will the same inflammation result

from a freely movable tube as from a naturally restricted one. The size and condition of the mastoid cells probably have a bearing upon the middle ear conditions where there is faulty ventilation of the tube.

We have found associated with chronic middle ear catarrh most frequently a tube which is swollen and more or less granular in outline. In color it is about that of the chronicly inflamed pharynx. Frequently, excessive mucous or mucopurulent secretion is found upon and within the tube. There is often hyperplasia of the adenoid tissue in Rosenmuller's fossæ. It is not rare to find adhesions in these fossæ. Excessively hypertrophied posterior ends of the inferior turbinates are often found associated with chronic salpingitis and middle ear catarrh, and I am convinced that hypertrophy of the posterior end of the turbinate plays a much more important part in causing eustachian insufficiency and its resulting otitis, than deformities in the anterior portion of the nose. In a number of cases we have found the hypertrophy so marked as to cover the opening to the tube, and very frequently the turbinate rests upon the anterior lip of the tube. The discharge of pus from the sphenoid and from the posterior ethmoid cells almost always flows over the middle turbinate and into the end of the eustachian tube. In these cases the mucous membrane covering the tube is always much swollen and has somewhat the appearance of raw beef.

Rarely we find a true atrophy of the membrane lining the tube. These cases form crusts, and the general appearance of the membrane is like that of the pharynx under similar conditions. In the few cases of atrophy we have been unable to obtain any lasting benefit from any line of treatment.

In treating the eustachian tube by the aid of the nasopharyngoscope we must, as in any other line of treatment, not forget the general condition of the patient, nor can we ignore the nasal and pharyngeal pathology. The great advance is in being able to treat more successfully the suppuration in the posterior nasal sinuses and in recognizing and removing more successfully pathologic growths in and about the tube.

It is of the greatest importance to use as little force as possible. Much care must be used both in making applications and in passing bougies through the tube. We have used with considerable success a solution of silver nitrate 10 per

cent about the pharyngeal end of the tube, but within the tube too much reaction may follow its use. Argyrol 40 per cent is much safer to use, and it can be carried upon a cotton tipped applicator or injected through the small eustachian syringe. In cases of true stricture within the tube much can be gained by passing cotton tipped bougies. In passing any instrument into the eustachian tube the tube should be constantly watched, for frequently the bougie or syringe tip catches in some fold of mucous membrane. If under these conditions continued pressure is persisted in, trauma is sure to result. Whenever the mucous membrane at the floor of the tube is seen to tighten or the bougie wire becomes flexed, it should be slightly withdrawn and another attempt made. Often by slightly rotating the wire the tip will pass by the obstruction. We have obtained the most marked results by passing the bougie every two days and by being careful not to pass too large a tip at any time. Slow dilatation without trauma may produce excellent results, but too rapid stretching producing trauma will almost always result in failure.

During the past eighteen months we have treated quite a large number of chronic middle ear catarrhs by treating the associated disease of the eustachian tube, but as many of them were treated at the same time we were studying and treating other conditions of the tube, there were not sufficiently frequent nor careful hearing tests made, and a report of many of them would be more or less indefinite. We therefore began the first of January to give our whole time to this class of cases, and have selected for treatment only such as have been of long standing and have received long courses of the usual treatment without benefit. By excluding cases of recent development, and not including those which have not received previous treatment, we have of course been working with severe and advanced types, and our results are not as marked when reviewed statistically, but when studied individually they give us some courage for further efforts with this condition.

For any who wish to more carefully study those cases I have recorded them in chart form, and for our present purpose I shall briefly review this chart. It may seem at first glance that the results as a whole are not particularly encouraging, but when we consider that only those cases were selected for study which were of long standing and which had received

long courses of treatment without benefit, it seems we have been able to accomplish much. This series of cases were all treated in the presence of my assistant, Dr. Cummings, at the Boston Dispensary, and the hearing tests were made when we were both present. By acting as checks upon each other, we feel that our findings are comparatively accurate.

There are in all eighteen cases. Thirteen are females and five are males. The youngest is 20 and the eldest is 65 years of age. Eight received benefit in hearing and ten practically none. Five of these were much improved in hearing. Fourteen suffered with tinnitus, and six were relieved.

One case (No. 7) was cured of severe attacks of dizziness, which were so severe as to make it impossible for her to go unattended upon the street.

No. 1, age 47, deafness for nine years; constant tinnitus, could hear voice in right ear 2 feet, in left ear 0. Could not hear whisper in either ear. At the end of ten weeks could hear voice in right ear 8 feet, and in the left 3 feet. The tinnitus was practically gone.

No. 4, age 31, increasing deafness for five years; almost constant roaring and rumbling. Had received long course of treatment without relief. We found a chronic secretive inflammation of tubes and a hypertrophy of the posterior ends of the lower turbinates and, treating the tubes for nine weeks, this patient was given sufficient hearing for all ordinary demands.

No. 7, age 49, shows more marked results than any of the others in this group. She had been deaf for several years and had grown rapidly so for two years. She suffered much from roaring and high pitched tinnitus. She had very frequent and severe attacks of dizziness. Examination showed moderate deflection of septum to left, general hypertrophic rhinitis, especially of the left lower turbinate. There were adhesions in right lateral fossa. Left lower turbinate rested upon tube. There was a general chronic hypertrophy of tubes. She could not hear spoken voice in right ear, and could hear it but at 6 feet in the left. She could hear whisper in left ear but 8 inches. The fork was, right ear 9/13, left ear 11/11. The adhesions were severed, the posterior end of turbinate removed, the tubes bougied and treated by argyrol, and she used an ointment of hydrastin muriate for eleven weeks. The

dizziness was gone at end of fourth week, there was no tinnitus after seventh week, and at the end of eleven weeks she heard spoken voice 18 feet in right ear and 20 feet in left. A whisper was heard 4 feet in right ear and 9 feet in the left. The fork was, right ear 31/11, and left ear 34/12.

No. 9, age 48, growing deaf over five years; suffered with roaring and at times high pitched tinnitus. Treated as private patient and at various clinics. She had much hypertrophy of right tube and considerable adenoid in left fossa. By curetting fossa and treating the tubes the tinnitus was much relieved and the hearing was much increased.

No. 12, age 46, deaf in left ear since infant; growing deaf in right for three years. Constant treatment of right ear for two years. We found a hypertrophic secretive salpingitis. The left ear was an old effusion, the right ear chronic catarrhal. The tinnitus was cured and the hearing more than doubled in the right ear.

No. 14, age 20, growing deaf four or five years. Throbbing and rumbling with a hissing tinnitus. Eighteen months' treatment by private physician, nine months at Eye and Ear Infirmary. The septum was deflected to left and there was a general hypertrophy of turbinates. Both tubes were patulous and constricted. By removing posterior ends of turbinates and treating the tubes the tinnitus subsided and the hearing was nearly trebled.

No. 17, aged 39, was given much relief in right ear by removing adenoid and treating epipharynx and tubes.

The other cases, as shown by chart, were not practically improved, and yet we could not discover any reason why they were not relieved while others were given marked relief. The results shown in these eighteen cases are practically the same as we have found in a large number of cases treated, and we have reported these because they were studied as a group and were, therefore, better followed and recorded. They were all severe and long standing cases, and if we can relieve even a comparatively small number, yet failing in all the others, it seems worth the effort.

CHART SHOWING RESULT UPON AURAL SY

Case	Age	Symptoms		Previous Treatment
		Deafness	Tinnitus	
1 Mrs. D.	47	Beginning 9 years ago has varied but generally progressed. Very bad for past 3 years. Weather first affected, but not now.	For first few years roaring and rumbling. Now hissing and bell tones.	Eight years ago treated constantly for 9 months: fi twice and then once a week. Off and on at different tim since. Past 6 months inflated every week. No appare change.
2 John J.	53	Growing deaf for number of years. Varied at first but not for 2 or 3 years. Father very deaf before 50.	For a number of years marked roaring and bell-like noises. None in several years past.	Nine years ago treated for 7 or 8 months. Was relieve Been treated past 2 years without relief.
3 Mrs. S.	44	Eight years ago, after severe cold, had attack of deafness lasting few weeks. Several other attacks lasting short periods. Steadily growing deaf for 2 years.	Very severe, high-pitched for about year. Throbbing at times.	Constant treatment for 18 months. Some nasal operati Catheter, massage.
4 H. S.	31	When child had attacks of deafness. Was free from ear trouble after 12 years till 5 years ago. Began with full feeling right ear. Later left ear began same symptoms. Deafness grown gradually worse.	Almost constant roaring and rumbling for 5 years. At times thumping, and occasionally high-pitched hissing.	Treated for 5 months at Eye and Ear infirmary 4 years ag Slight improvement. Three years ago treated by priva physician for 3 months, 3 times a week. Past 4 mont treated at Dispensary without improvement.
5 Mrs. W.	27	Growing gradually deaf for 5 years. Abscess in left ear when 14 years old. Mother and one brother deaf.	Hissing all of the time. Very severe at times.	At intervals for 4 years. Politzer. Catheter. Operati nose.
6 Wm. R.	40	Deafness at times for 10 years. Constant, and growing gradually worse for 6 years.	Hissing, roaring, and bell-like noises most of the time.	Been treated by several aurists for long periods. Past years treated at hospital.
7 Miss G.	49	Growing deaf for several years. For 2 years very deaf right ear, and rapidly growing deaf left. Frequent attacks of intense dizziness. Has fallen a number of times.	Roaring pulsation for several years. High-pitched at times.	Treated by inflation for nearly year, 3 years ago. Be treated by inflation past 7 months.
8 Mrs. D.	34	Begun growing deaf when in school. Grown gradually worse. Mother deaf at 30.	Some years ago much troubled by various head noises. None for several years.	Treated by various aurists in Boston and New York. Be treated at Boston City Hospital for past 10 months.
9 Mrs. J. B.	48	Deaf right ear 7 or 8 years, growing worse. Increasing deafness left ear for 5 years. Full feeling both.	Usually roaring. At times high pitched and very distressing.	Been treated at various hospitals. Treated for year and half by specialist as private patient.
10 Miss S.	25	Right ear deaf about year and a half. Left ear deaf for number of years.	Pounding and roaring in right ear.	Several operations upon nose. Two operations for ad noids. Inflated by catheter 3 times a week for over a yea
11 J. S.	43	Gradually becoming deaf for several years. Cannot hear anything except loud noises.	Very distressing, high pitched.	Off and on for number of years. Used sprays and Politz ing.
12 Anne K.	46	Right growing deaf 3 years. Deafness intermittent. Left deaf since infant.	Right ear very marked at times.	Right ear treated constantly for over 2 years, but h grown worse.
13 Mrs. B.	58	Deaf for number of years. Has grown worse during past 3 or 4 years.	None for several years.	Treated by catheter and painting throat for nearly 2 year
14 L. G.	20	Growing deaf both ears for 4 or 5 years. At times very bad.	Rumbling and throbbing most of time. Hissing at times.	Year and a half by private physi Nine months at E and Ear Infirmary.
15 Mrs. W.	33	Quite hard to hear for 3 years. At times worse. Very bad for year.	Occasionally much pressure.	About a year ago had 3 months' treatment. Was treate twice each week.
16 Mrs. H.	41	Right ear deaf 2 years. Left ear deaf 20 years. Growing worse in right.	Right ear severe ringing.	Most of time for 2 years.
17 M. G	39	Deaf in right ear for 5 years. Growing deaf in left ear for 3 years.	Intense roaring and ringing for 2 years.	Six months catheter at Boston City Hospital. Five month at the Boston Dispensary. No improvement.
18 Mrs. B.	63	Gradually growing deaf for several years.	Never.	Treated by several aurists and at a number of hospitals

AURAL SYMPTOMS, IN CHRONIC MIDDLE EAR INFLAMMATION, BY TREATMENT OF THE EUST

MENT	EXAMINATION				R.
	General	*Nose*	*Eustachian Tube*	*Membrana Tympani*	
tly for 9 months: first nd on at different times ery week. No apparent	Well nourished and preserved. Says except for catarrh back of nose is well.	Slight deflection of septum to the left. Moderate hypertrophy of the lower turbinates.	Adenoid in both fossæ. Chronic secretive inflammation of both tubes. Small bougie passed with difficulty in left.	Right retracted and thickened. Left much retracted, calcerous deposit in posterior quadrant.	2' 0 0 12/16
months. Was relieved. t relief.	Very fat and flabby. No history of illness. Tobacco and alcohol to excess.	Very turgescent mucous membranes. Deflected septum and ridge on the right side.	Much swollen mucous membranes. Muco-purulent secretion both tubes.	Right thick and retracted.	4' 0 0 12/7
. Some nasal operation.	Good.	Left middle turbinate gone. Some pus and several polyps in posterior ethmoid cells.	Considerable adenoid in both fossæ. Much swollen membranes. Considerable secretion in tubes.	Right retracted producing considerable tilting of malleus. Left retracted to promontory.	2' 0 0 9/16
ar Infirmary 4 years ago. s ago treated by private a week. Past 4 months nprovement.	Excellent.	Posterior ends of turbinates much hypertrophied.	A thick tenacious mucous covers a chronic hypertrophied tube. Movement restricted by turbinates. Much constriction at isthmus in left.	Both retracted but fairly movable with Siegler's otoscope.	5' 1 0 10/16
er. Catheter. Operation	Normal.	Small perforation in septum from sub-mucous operation. Polyp left side attached to the middle turbinate.	Considerable central adenoid restricting movements of both tubes. Narrow isthmus both sides.	Right thick, retracted movable membrane. Left calcerous crescent in lower quadrant.	6' 2' + 16/15
for long periods. Past 3	Good physique and excellent health.	Portion of right middle turbinate gone. Lower turbinates show cicatrices from cautery.	Both tubes contain muco-purulent secretion.	Both very thick and much retracted.	0 0 0 9/11
year, 3 years ago. Been ha.	Pulse normal. Eyes normal. Urine normal.	Moderate deflection to left. Hypertrophy right turbinate. General hypertrophic rhinitis.	Adhesions in right fossa. General hypertrophy. Left lower turbinate rests on tube.	Right much retracted and injected. Left much retracted.	0 0 0 9/15
ston and New York. Been for past 10 months.	Anæmic and rather frail. Heart and lungs normal.	Anæmic mucous membranes. Puffy turbinates.	Anæmic. Edematous in appearance. Very narrow isthmus in both tubes.	Very thin. Slight retraction of right. Much retraction of left.	2' 0 0 14/15
als. Treated for year and a tient.	Fairly good.	General hypertrophy. Ridge left side of septum.	Right hypertrophic. Left much adenoid in fossa.	Both thickened. Moderate retraction.	1' 0 0 11/8
. Two operations for adeoses a week for over a year.	Not robust. Rather weak pulse.	Cartilage resected. Left middle turbinate gone. Right middle turbinate hypertrophied posteriorly.	Adenoid in fossæ. Tubes much swollen. Crusts of muco-pus in both tubes.	Both very much thickened and retracted.	5' 0 0 17/11
s. Used sprays and Politzer	Good.	Deflection to right. Moderate hypertrophy of turbinates.	Bands both fossæ. General hypertrophy. Much restriction of movement. Marked stricture in right.	Thin, much retracted membrane both sides.	0 0 0 7/16
for over 2 years, but has	Very thin, but physically normal.	Normal.	Hypertrophic right tube, purulent left.	Right much retracted. Left very cicatricial.	9' 2' + 16/21
ng throat for nearly 2 years.	Systolic murmur. Chronic nephritis.	Purulent ethmoiditis left side. Left antrum filled with pus.	Purulent epi-pharynx and tubes.	Right very thick. Left calcerous deposit.	3' 0 0 13/6
. Nine months at Rye	Apparently well.	Septum to left. Ridge left side. Turbinates hypertrophied.	Very patulous. Stricture both tubes. Considerable secretion in lateral fossæ.	Both membranes retracted.	14' 2' 0 15/18
' treatment. Was treated	Excellent.	Thick septum. Large lower turbinate.	Chronic granular mucous membrane. Considerable adenoid in both fossæ.	Thick and retracted.	6' 2' + 14/17
	Fairly well.	Practically normal.	Much adenoid. Very swollen tube. Much secretion.	Right much retracted. Left very cicatricial.	8' 3' 0 15/15
n City Hospital. Five months improvement.	Very excitable. Otherwise well.	Sub-mucous operation 4 years ago. Pus in posterior ethmoid cells, left side.	Marked hypertrophy. Pus in epi-pharynx and over tubes.	Both retracted to promontory.	2' 0 0 5/16
t at a number of hospitals.	Rather frail. Pulse irregular	Very pale membranes. Septum moderately deflected to right.	Membranes pale. Restricted movement.	Practically normal.	3' 0 0 11/8

			Treatment	Results		
pani	R. Hearing L.			R. Hearing L.		Tinnitus
t thick- ructed, n poste-	2′ Voice 0 0 Whisper 0 0 Low fork 0 12/16 C fork 9/19		Curetted both lateral fossæ. Passed bougie every 2 or 3 days for 9 times. Applied Argyrol with syringe twice a week for 10 weeks.	8′ Voice 3′ 7″ Whisper 2″ + Low fork 0 20/16 C fork 20/18		Practically gone.
ructed.	4′ Voice 5′ 0 Whisper 0 0 Low fork 0 13/7 C fork 11/7		Argyrol to tubes. Ag. No. 3 to epi-pharynx. Soluble iodin gtt. viii t. i. d. Protargol spray. Treated 10 weeks. No apparent change.	5′ Voice 5′ 0 Whisper 0 0 Low fork 0 11/3 C fork 12/7		
ducing of mal- ted to	3′ Voice 2′ 0 Whisper 0 0 Low fork 0 6/18 C fork 6/21		Curetted polyps from ethmoid. Cleared lateral fossæ. Bougied tubes. Applied argyrol. Prescribed thigenol ointment to use twice a day. Treated 11 weeks.	No improvement.		Much less hissing. No throbbing.
fairly iegler's	5′ Voice 3′ 1 Whisper 6″ 0 Low fork 7 19/16 C fork 14/18		Removed posterior ends of turbinates. Right bougied but once. Left 6 times. Argyrol to tubes twice a week for 9 weeks. Ag. No. 3 to vault of pharynx.	20′ Voice 12′ 6 Whisper 3′ + Low fork + 35/15 C fork 22/16		Only occasional hissing.
tracted e. Left n lower	6′ Voice 0 2′ Whisper 0 + Low fork 0 16/15 C fork 7/17		Removed polyps. Curetted adenoid. Passed bougie 5 times. Argyrol 9 weeks.	No improvement.		No improvement.
d much	0 Voice 1′ 0 Whisper 0 0 Low fork 0 0/11 C fork 13/16		Bougied twice, passed fairly easy. Argyrol to tube for 6 weeks. Secretion much less.	No improvement.		Less noise.
ed and ch re-	0 Voice 6′ 0 Whisper 8″ 0 Low fork + 9/13 C fork 11/11		Separated bands right fossa. Removed posterior end left turbinate. Bougied both tubes 3 times. Argyrol to tube and into right mid-ear. Gave Hydrastin ointment. Treated 11 weeks.	18′ Voice 20′ 4′ Whisper 7′ + Low fork + 31/11 C fork 34/12		No noise for 4 weeks. No dizziness for 10 weeks.
traction ction of	2′ Voice 0 0 Whisper 0 0 Low fork 0 14/15 C fork 9/21		Bougied tubes 11 times. Argyrol to tube. (Iron as. and strychnine internally.) Treated 8 weeks.	4′ Voice 1′ 2′ Whisper 0 0 Low fork 0 20/19 C fork 14/18		
oderate	1′ Voice 1′ 9′ 0 Whisper 6″ 0 Low fork 0 11/8 C fork 13/9		Curetted left fossa. Passed bougie 5 times. Argyrol to both tubes 3 times a week for 5 weeks.	8′ Voice 15′ 2′ 5′ Whisper 4′ 8″ + Low fork 0 24/8 C fork 20/8		Much relieved.
ickened	5′ Voice 1′ 0 Whisper 0 0 Low fork 0 17/11 C fork 9/19		Removed adenoid from crest and fossæ. Bougied both tubes. Applied argyrol. Gave Hydrastin muriate ointment for nose, and soluble Iodin 8 drops before meals. Treated 11 weeks.	Practically same as at examination.		No pounding. Only occasional roaring.
ed mem-	0 Voice 0 0 Whisper 0 0 Low fork 0 7/18 C fork 5/19		Removed bands. Bougied tubes. Applied Argyrol. Treated 12 weeks.	No improvement.		No improvement.
ed. Left	9′ Voice 2′ 2′ Whisper 3″ + Low fork 0 18/21 C fork 5/20		Argyrol to tube every 2 days. Hydrastin ointment morning and night in nose and epi-pharynx. Much improved.	20′ Voice 2′ 8′ Whisper 4″ + Low fork 0 30/15 C fork 5/21		Gone for 3 weeks.
Left cal-	3′ Voice 4 1/2 0 Whisper 0 0 Low fork 0 13/8 C fork 16/7		Removed left middle turbinate and ethmoid cells. Opened antrum. Gave thigenol. Applied argyrol to tubes. Treated 10 weeks. Nose and epi-pharynx much improved.	No change.		
tracted.	14′ Voice 8′ 2′ Whisper 6″ 0 Low fork 0 15/16 C fork 8/22		Bougied every 2 days for 2 weeks. Argyrol to tubes. Posterior ends of turbinales removed. Treated 11 weeks.	20′ Voice 20′ 9′ Whisper 5′ + Low fork + 42/16 C fork 34/17		Gone.
ed.	6′ Voice 5′ 2′ Whisper 4′ + Low fork 0 14/17 C fork 9/18		Removed adenoid from fossæ and curetted adenoid granulation from tubes. Treated with argyrol for 9 weeks. Thinks hearing is much better.	No change.		Much less pressure.
ted. Left	8′ Voice 0 3″ Whisper 0 0 Low fork + 15/15 C fork 16/16		Removed adenoid. Ag. No. 3 to epi-pharynx. Argyrol to tubes every 3 days for 9 weeks.	13′ Voice 0 18″ Whisper 0 + Low fork 0 24/15 C fork 17/18		No ringing.
grannon-	2′ Voice 5′ 0 Whisper 8″ 0 Low fork 0 5/14 C fork 8/15		Operated on left ethmoid. Argyrol to tube. Ag. No. 3 to pharynx. Marked improvement at end of 10 weeks. Treated 14 weeks.	20′ Voice 12′ 7′ Whisper 18″ 0 Low fork + 31/15 C fork 14/16		Gone.
i.	3′ Voice 4′ 0 Whisper 0 0 Low fork 0 11/6 C fork 14/7		Argyrol to tube twice a week for 11 weeks. Hydrastin ointment in nose.	3′ Voice 3′ 0 Whisper 0 0 Low fork 0 10/9 C fork 11/15		

XLV.

MIDDLE EAR SUPPURATION AND LIFE INSURANCE.

By Emil Amberg, M. D.,

Detroit.

Concerning life insurance examination and middle ear suppuration, we must distinguish between an acute and a chronic process. The acute process admits but one decision, and as such deserves no further consideration. In chronic suppurative cases we must distinguish between ears which have been operated upon and ears which have not been operated upon.

In regard to the first cases, I call attention to the article by Professor Friedrich Roepke.[1] I only quote the following: "Which cases must be considered cured? Certainly all cases in which the whole osseous cavity is entirely covered by epithelium. But I do not hesitate to consider from the standpoint of the life insurance companies those cases cured in which in the region of the ostium of the tube an epidermization has not taken place, but in which the cavity is otherwise smooth and open to inspection. The secretion of mucus from the tube, which in these cases easily occurs, according to our experiences, is of no significance for the life expectancy of the individual under consideration, as we have almost exclusively to deal with people who belong to the better class," etc.

In the Austrian Otological Society, April 3, 1911,[2] F. Alt's suggestion was accepted unanimously, namely, that the chief physicians of the life insurance companies should be informed that such people should not be excluded from life insurance on whom the radical operation has been suitably performed, and whose middle ear cavities have been epidermized. An eventually still existing secretion from the tube should not be a contraindication for acceptance. Ruttin remarks that the localization cannot be restricted to the tube, because one knows that the socalled mucous membrane cures are just as favora-

ble. V. Urbantschitsch succeeded in having radically operated persons insured with a very small increase of the premium. Frey is of the opinion that a radically operated ear is safer than a normal ear, because the danger of an acute otitis is excluded.

In illustration of the second class of cases, namely, of those which have not been operated upon, I report the following in which I was asked for my opinion:

Mr. J. G. P., 41 years old, married, consulted me about his ear in relation to a life insurance examination. Mr. P. told me that about thirty years ago a kernel of wheat had entered his right ear and that the ear does not discharge at times for four or five months, also that the discharge is never profuse. He says that the discharge was at times a little offensive, but not to speak of. He had never any pain around the right ear, was never dizzy, and had never any noises in the ear.

The right ear, the ear in question, shows a destruction of the drum membrane.

On November 1st I put a piece of cotton in his right ear, which he accidentally left in the ear until November 9th. The cotton was practically dry. On November 9th I applied some powder (xeroform) to the middle ear. On November 11th this powder had disappeared, but I did not detect any free discharge. November 1st the hearing test in the right ear revealed the following: a^1 fork heard by bone and air conduction, fork with 96 and with 128 vibrations, heard by bone but not by air conduction.

A fork of 20 vibrations was not heard by air conduction in his right ear, but a fork of 40 vibrations was heard, also one of 512, 1024, 2048, 4096 vibrations. The Galton whistle was heard at 0.0, i. e., very high tones were heard. On November 11th Politzer acoumeter was heard in a distance of 7.5 inches in the right ear. A watch in a distance of 1 inch in the right ear (a watch ordinarily heard more than 100 inches away).

REMARKS.

There exists a destruction of the drum membrane in one of the ears, the right ear. The question arises: Has the applicant a dangerous ear? On page 134 of Kopetzky's Surgery of the Ear, we find two figures, No. 28 and 29, which

show the nondangerous type of perforation of the drum membrane. The applicant's ear shows the picture between Fig. 28 and 29, i. e., inasmuch as the head of the stapes is visible, but the perforation is larger than in Fig. 29 and smaller than in 28.

Kopetzky (page 135) says: "Generally speaking, there is no danger of intracranial complication in this class of chronic middle ear suppuration, which we have designated, for want of a better name, as nondangerous."

If the applicant should ask me whether I would advise an operation for his ear, the socalled radical operation, I would advise against it, because I would not feel justified in performing it, enforcing my opinion by that of Politzer. In this case a suppuration of the antrum could not be diagnosed. I attribute the secretion of which the applicant speaks to the eustachian tube, and of course an affection of the nose may aggravate it. It probably disappears with the nose affection, as it has done for thirty years. The applicant told me that he was admitted.

Heine, in his book "Operations on the Ear," second edition, 1906, page 66, says: "We are accustomed to distinguish between dangerous and nondangerous chronic middle ear suppurations. The location of the perforation in the drum membrane and the kind of suppuration tells us to which category a certain suppuration belongs." A marginal perforation is classed by him as dangerous, as a suppuration of the bone. He considers a perforation marginal even when perhaps only a narrow border of its anterior portion, or practically nothing, has been left. The presence of free annulus tympanicus in the region described is considered characteristic.

Holger Mygind[3] considers cases of chronic suppuration with a central perforation comparatively nondangerous, whereas cases with a marginal perforation are much more dangerous, especially if there is little pus, and if it is mixed with cholesteatomatous material. He thinks that cases with a dry perforation and those which are dry after a radical operation should be admitted without question.

From the chief medical department of two of the largest life insurance companies in this country I have received the following answers (in part):

1. "This company does not accept anyone showing middle

ear suppuration, with rare exceptions. After the suppuration has ceased, and sufficient time has elapsed to show that it is probably a permanent cure, we are willing to take them at ordinary rates unless totally deaf."

2. "I beg to say that our statistics are so meager in this respect, I fear they could not be of any assistance to you. Where there is a purulent otorrhea, we refuse to take the risk at all, until assured that a complete cure has been effected."

CONCLUSIONS.

The relation of chronic middle ear suppuration to life insurance is by no means a simple question. Concerning radically operated cases, the decision is not very difficult. Nonoperated cases, however, require careful discrimination. It would seem that not all people with a chronic middle ear suppuration should be excluded from life insurance, even if they have not been operated upon.

The unilateral or bilateral diminution of hearing also enters into consideration.

REFERENCES.

1. Roepke, Friedrich: Handbook of Special Surgery of the Ear and Upper Respiratory Tract, p. 349 See also Medical Record, April 13, 1912, pp. 735 and 736.

2. Alt, F.: Archiv. fuer Ohrenheilkunde, Vol. 88, Parts 1 and 2, pp. 48 and 49.

3. Mygind, Holger: Zeitschrift für Ohrenheilkunde, Vol. 64, part 4, p. 379.

XLVI.

THE RELIEF OF TINNITUS BY THE USE OF NITRATE OF SILVER APPLIED WITHIN THE EUSTACHIAN TUBE.

By William C. Braislin, M. D.,

Brooklyn.

The systematic application of nitrate of silver within the eustachian tube, as employed by me during the past year, has seemed of great benefit, particularly in tinnitus due to swelling and engorgement of the mucous membrane lining the tube. This is recommended as an addition to the treatment by the catheter and other methods.

Incidentally I may refer to some other means which I have employed to relieve congestion by the tube. Thus I have occasionally used during the last four years a small extensible knife which I had fitted into the lumen of a catheter-shaped canula, by means of which blood letting from the mucous membrane of the mouth of the tube has been performed with beneficial results in some cases. Likewise, I have employed the method of breaking up adhesions about the mouth of the tube with the finger, as recommended by Dr. Logan at the meeting of this Society in 1909. Injections of solutions into the tube with the flexible stiletted bougie have also been used. During the past winter I have systematically employed the applications of silver in cases of tubal catarrh, O. M. C. C., and mixed middle and internal ear cases; in short, in all classes of nonsuppurative ear cases in which swelling of the tube was an evident accompaniment of the other conditions present. For the most part these applications have been made by a small cotton pledget, twisted on a flexible wire applicator, dipped in the silver solution and carried through the lumen of the catheter into the eustachian tube for the distance of a quarter of an inch to one inch. This is done at the finish of the ordinary catheter inflation and be-

fore removing the catheter from its position. For greater convenience, previous to the beginning of inflation through the catheter, the wire is wrapped and laid within easy reach beside the bottle of solution. On finishing the inflation the wire is dipped into the solution and carried through the lumen of the catheter into the eustachian tube, the patient meanwhile swallowing, if necessary, to facilitate its easy passage. The application made, the wire is at once withdrawn. A 5 per cent solution of nitrate of silver is usually employed, which, when acute inflammation is present in the tube, creates a sharp, but only temporary, sensation of pain.

Instead of appending here a considerable series of cases of the various ear diseases referred to above, treated by the method of combined inflation and nitrate of silver applications within the tube, I decided that a few summarizing statements would better economize time and serve the purpose of this communication equally well. I have proved to my own satisfaction that the relief of tinnitus is more rapid and effectual where silver applications within the tube are employed. Several recurrent or chronic cases so treated have been under observation and occasional treatment for from three to ten years. In these cases the patient generally obtained greater relief in the same time than formerly. Tubal swelling is nearly always accompanied by tinnitus, and it is a relief to the patient to get improvement from tinnitus of tubal origin, even should other symptoms be unrelieved.

No result is expected by this method in the treatment of tinnitus due to immobility of the stapes, from tinnitus due to labyrinthine or auditory nerve lesion, or to that of the nervous centers. Tinnitus due to digestive, circulatory or general diseases is not appropriately treated alone by local method, but congestion in and about the tube is in part relieved by local applications.

In nearly all of these cases information as to the actual conditions about the mouth of the tube have been derived from the pharyngoscope, chiefly, however, in detecting some causes of tinnitus which might possibly have been overlooked, such as posterior hypertrophies of the turbinates, small polypi impinging against the mouth of the tube, specific ulcers and others.

Tinnitus which is due to inflammatory conditions of the

tube, seems, to the writer, to yield more rapidly and permanently when nitrate of silver applications within the tube are added to the other methods of treatment. The presence of diseases of the tube must be regarded as an indication or tendency on the part of the sufferer to acquire more serious ear lesions. All contradictory influences in the nose and pharynx are to be eradicated as stringently as in ear diseases of a more advanced character; otherwise, recurrence will be proportionally earlier and more severe.

The applications of silver within the tube have the advantage over other and more elaborate methods of treating the tube in the comparatively slight additional time required to apply the treatment; and the writer believes its usefulness to be superior to the use of the plain or unmedicated bougie. Its systematic application in his own experience has proved more generally useful than the use of the metal bougie for carrying the electric current.

XLVII.

HISTORY OF A CASE OF CHOLESTEATOMA OF THE EAR.

By Emil Gruening, M. D.,

New York.

The patient of whom I wish to speak was under my observation thirty years. She was a woman who had come to me when she was 24 years of age. In her infancy and youth she had often been troubled with a discharge from the right ear. At the time of the first examination, thirty years ago, the drum head did not exist; there was neither a malleus nor an incus; the tympanic cavity was dry and epidermized, the aditus ad antrum communicated with the middle ear as freely as if a radical operation had been performed. No actual operation had, however, been done. The process of disease had formed a spacious cavity from which now and then cholesteatomatous material descended into the external auditory canal. The ear was absolutely deaf. The patient would at times, especially after immersion of the head in a warm bath, complain of dizziness. I ascribed this symptom to the swelling of the cholesteatoma from imbibition of water, and treated the ear, when these symptoms appeared, by the instillation of glycerin and alcohol. After a few days of such treatment the cholesteatomatous material became dry, appeared in the canal, and the head symptoms would then disappear. When the attention of the patient was drawn to the circumstance that the water-logged condition of the cholesteatoma might cause the head symptoms, and she refrained from filling the ear with water, the symptoms mentioned appeared but rarely.

During this long period of observation she was also seen, during her trips to Europe, by Politzer of Vienna, Bezold of Munich, and other noted aurists. None of these men proposed an operation, and all agreed that under the given conditions the treatment which she followed under my direction was sufficient.

In the month of March of this year she began to suffer from copious uterine hemorrhages. A gynecologist, who was consulted, found that she suffered from uterine fibroids, and advised that the uterus be removed, but also stated that an immediate operation was not imperative.

On March 23d of this year she was suddenly taken with a severe chill, high temperature (104.6), and frequent vomiting. The family physician, who knew that she had been under my observation for her aural disease, considered the possibility of a systemic infection by way of the lateral sinus, and desired to meet me in consultation. I saw her in the evening of that day. While I was in her house she had a severe chill and vomited. The temperature had risen to 105°. The examination of the ear showed what I had seen before, viz., a perfectly dry middle ear, no tenderness of the mastoid and postmastoid regions, no enlarged glands, and no sensitiveness along the course of the jugular vein. The ophthalmoscopic examination was negative, and I came to the conclusion that the sepsis from which the patient suffered was not caused by the condition of the ear.

In order to observe the patient more fully, and to have the necessary laboratory tests made, she was transferred to a general hospital that very evening. The leucocyte count was 24,400, with 88 per cent polynuclears. Enough blood was taken from the veins of the arm to make a blood culture. The gynecologist who had seen her previously, reexamined her at the hospital that evening, and came to the conclusion that the sepsis was not due to the uterus. He found, however, some tenderness of one ovary, and ordered the application of an ice bag. During the night the patient vomited frequently, her temperature remained high, and she had several severe chills. The mind remained clear.

On the following morning, March 24th, I met Dr. Whiting in consultation, who corroborated my views of the case. One of the gynecologists of the hospital, who was called in consultation disagreed with the previous finding in regard to the uterus, and proposed an immediate panhysterectomy. The husband of the patient consented. I was present at the operation. On cutting through the right ovarian vein a small amount of pus escaped. The removed uterus contained several submucous and intramural fibroids. The center of some of these fibroids were softened and discolored. The patient

rallied from this operation, but the temperature rose to 105.6° and the chills and vomiting continued.

On the morning of the 25th of March we received the laboratory report of the blood, which had been made under Dr. Libman's supervision. The streptococcus was found. A medical consultant, who was called by the family, advised an exploration of the right lateral sinus with ligation of the jugular. The patient had 40 respirations per minute, pulse of 140, and in view of the fact that the hemoglobin was 60 per cent, he thought she would bear the operation well. In order to gain time a surgeon ligated the jugular while I laid bare the sinus. Though the operation lasted only a few minutes the patient collapsed, but rallied after an intravenous infusion of hot saline solution. No thrombus was found in the sinus. The patient died that same evening. Dr. Libman made a local postmortem examination of the region of the lateral sinus, but found no clot. The pathologist of the hospital examined the uterus microscopically and found purulent thrombi in the uterine veins.

This case demonstrates that though a chronic disease of the middle ear may be associated with a systemic infection and a positive blood culture, we are not entitled, in the absence of local symptoms, to assume that the diseased ear must necessarily be the causative factor. We should exclude all other possible foci of infection. The result of the postmortem inspection of the sinus and the microscopic examination of the uterus showed that the operative procedure on the sinus was unnecessary.

XLVIII.

A CASE OF PARACOUSTIC VERTIGO AND NYSTAGMUS.*

(PRELIMINARY REPORT.)

By JOHN RANDOLPH PAGE, M. D.,

NEW YORK.

Without reference to the literature on the subject, I wish at this time to report the history and examinations of this unusual case in order to hear the opinions of the gentlemen present tonight concerning it.

The patient is a well nourished man 44 years of age, of powerful build, six feet four inches tall, and weighs 235 pounds. He has led an outdoor life of great physical activity, and except for disturbance due to nose and throat trouble has been practically free from illness.

There is no hereditary history of deafness, or disease. He states that as a child he slept with his mouth open, and he had frequent earaches until he was twenty-two years of age, but he never remembers having had a discharge from either ear. He was subject to attacks of tonsillitis, and when a boy had a swelling in the right side of his neck which persisted for several months. At present his neck is tender but there is no enlargement of the glands.

Deafness and hissing sounds have been present in right ear since boyhood, with more or less feeling of stuffiness, and the deafness has increased of late. Until a year ago he could relieve both the deafness and the stuffiness by dropping and twisting his jaw, and although this no longer affords him the relief it formerly did, he continues to do it in conversation from habit.

For the past seven years any exertion that increases the action of his heart, such as lifting, running, or even laughing

*Read before the New York Academy of Medicine, Section on Otology, April 12, 1912.

heartily, makes him dizzy and causes him to stagger, and at the same time the objects seen seem to move up and down with the pulsations of his heart. He first noticed this after running up a mountain side to get a shot at some wild turkeys. When he reached the place he wished to shoot from, he found he had to wait several minutes before the object, or rather his heart, steadied itself sufficiently for him to take aim.

Since November, 1910, certain sounds seem to almost blind him, and cause him to lose his balance to such a degree that he will fall if he does not catch hold of something for support. He can determine no definite direction in his tendency to fall. When the disturbance is sufficient to cause a loss of equilibrium, he is confused and "blinded" by the movement of the objects in front of him (the "shattering," he expresses it), and he feels like sinking to the ground. These sensations are relieved when the pitch is either raised or lowered beyond a certain point. Hammering on boilers and riveting machines, as well as whistles, produce the same disturbance. He had the pitch of the steam whistle on his plant lowered so that it does not now disturb him to the extent it did before.

AURAL EXAMINATION AND FUNCTIONAL TEST.

October 6, 1911. Right drum membrane: Dull gray in color, markedly retracted and lusterless.

Left drum membrane: Dull gray in color, considerably retracted and lusterless. Weber: Lateralizes to right ear most positively when fork is placed on vertex, and still refers the sound to that ear when the fork is placed on the left mastoid. Rinne: Positive for both ears. Acoumeter: Right ear, 2 inches; left ear, 6 inches. Whisper: Right ear, 18 inches; left ear, 15 feet. Lower tone limit: Both ears practically normal; right ear, hears 16 fork indistinctly; left ear, hears 16 fork distinctly. Upper tone limit: Right ear, 6.00 Galton whistle; left ear, 2.00 Galton whistle.

EXAMINATION OF NOSE AND THROAT.

Patient is suffering from cold in head. Turbinates are swollen, and there is a large spur on the right side of the septum. Tonsils are not enlarged, but they are inflamed, and the whole mucous membrane of the nose and throat is swollen.

Eustachian tubes: Right admitted no air on catheterization; left admitted air fairly well.

October 7, 1911. Right ear: Pipe sounds below a^3 (1740 vibrations) produce little or no effect on the vision, or the eyes themselves. At this pitch, however, the disturbance begins and continues throughout the whole octave, until a^4 (3480 vibrations) is reached, above which the disturbance ceases, the maximum disturbance being caused by sounds in the middle of the octave.

He states that at the beginning of the sound the object he is looking at moves downward and to the left, and remains in that position with a wavering movement until the sound ceases, it then moves back to its original position. The eyes rise and fall when the sound is produced and stopped, and when the most effective sound is prolonged, equilibrium is disturbed.

The nystagmus following rotation forward with the anterior vertical canal (and necessarily the posterior of the opposite side) placed horizontally, first on one side and then on the other, is equal (about sixteen seconds). Rotation backward, the same, nystagmus lasting about twenty seconds. When lying down the effect of sound is the same as it is when standing, that is, the eyes move in the same direction, namely, in the plane of the right anterior vertical semicircular canal.

After the above examination was made the patient returned home until the 11th of November, when he was admitted to the Manhattan Eye, Ear and Throat Hospital for observation.

For the relief of his vertigo a lumbar puncture was performed and twenty cubic centimeters of normal cerebrospinal fluid were withdrawn, and at the same time, because of the marked retraction of the right membrana tympani and the closed condition of the right eustachian tube, a free myringotomy was done. Intense headache, dizziness and nausea followed, and persisted for the next forty-eight hours, and for two weeks the slightest exertion in the upright position caused severe headache and dizziness.

Despite the immediate discomfort from the lumbar puncture, he declared that for the first few days after the myringotomy the hearing in the right ear was much improved, and that there was a grateful feeling of clearness in it. The incision in the drum soon healed, however, and the good effect was lost. Two more myringotomies were performed, and relief followed only

until the incision closed, so a crucial incision was then made in the membrane, and the edges of it were folded in with the hope that more permanent benefit would be obtained, but in this we were disappointed, for although the incision remained open the inflammation which followed in the middle ear offset the good that might have been obtained from the ventilation of it. After three weeks the patient left the hospital with a slight discharge from the right ear. His vertigo was much improved, and a marked change in the effect produced by sounds was noticed. Sounds that before would have made him fall had no effect on him.

The following is a report of the examination on the 9th of February, three months after the lumbar puncture was performed:

The staggering tendency has entirely gone. No dizziness at all. Right ear and side of head is very sensitive to the sound of his own voice, and the friction of his hand on his ear. This was immediately relieved by the removal of a crust which had formed over the opening, now healed, in the drum membrane.

Tinnitus remains the same. Sounds of lower pitch than before cause objects to move in front of him, and now his own voice produces the same effect if he speaks loudly, or laughs in a high key.

High tones (whistles), which formerly were troublesome, are no longer so, but lower pitched ones are. He has had to raise the pitch of the steam whistles on his plant back to what they were formerly.

In speaking, the pronunciation by him of certain letters and words produce marked effect. The eyes can be seen to move upward and to the right, and he says the object he is looking at moves downward and to the left, when he pronounces the letters E, P, T, Z, etc., or such a sentence as "Ease it off." The same effect is more markedly produced when he whistles certain notes, and if these sounds are prolonged his equilibrium is upset.

It was also found at this examination that when the note d^3 was sounded on a pipe the eyes moved straight upward, and he said the object in front of him moved straight downward. Notes of higher pitch caused the eyes to move upward and to the right, and those of lower pitch caused them to move downward and to the left.

In addition to the sensitiveness of the right vestibular apparatus to certain sounds, it is interesting to note that this is a case of deafness in which marked temporary improvement in hearing was obtained by ventilation of the middle ear, first, by habitual movement of the jaw to open the tube, and later by myringotomy, and in which the early history points to middle ear deafness, yet according to the classical tests it is one of nerve deafness, the low tone limits being normal, Rinne positive, and the upper tone limit on the right side lowered to 6.00 Galton whistle. Despite the positive Rinne, however, in the Weber test the sound is positively referred to the poorer ear.

XLIX.

PRIMARY LATERAL SINUS THROMBOSIS WITHOUT INVOLVEMENT OF THE MASTOID PROCESS OR TYMPANUM.*

By H. L. Lynah, M. D.,

New York.

Case.—M. W., female child, 4 years of age, admitted to the Kingston Avenue Hospital January 16, 1910, suffering with a severe type of postnasal and pharyngeal diphtheria. On physical examination the child was well nourished. Eyes, normal; ears, both membranæ tympanorum normal; nose, obstructed with a thin sanguineous discharge; throat, exudate on both tonsils, pillars of fauces, uvula, postpharyngeal wall, with almost complete obstruction of the nasopharynx. There was marked cervical cellulitis with a collar of brawny swelling about the neck. Heart, action rapid, regular, no murmurs. Lungs, negative. Antitoxin, 30,000 units, one dose; subcutaneous. There was but slight reaction. The neck was poulticed.

The following day, January 17th, the little patient was holding her own, and by the fourth day, January 20th, the temperature had reached normal. The lesion in the throat was entirely free from exudate, though there was a deep necrotic area where the right tonsil had sloughed out. The throat was constantly swabbed with argyrol and irrigated with saline solution. The cervical cellulitis had almost entirely disappeared from the left side, while on the right side the swelling had become more marked and was pointing as a cervical abscess just below the angle of the jaw. This abscess was incised and about two drams of pus evacuated, cultures and smears of which showed streptococcus. A rubber drainage tube was introduced and a wet dressing applied about the tube. The cavity was irrigated every two hours with normal saline solution.

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*Read before the Section on Otology, New York Academy of Medicine, May 10, 1912.

On the morning of the 21st, the fifth day after admission, the temperature rose to 101.4°, but fell to normal by midday, only to rise again in the evening to 102.4°. The cervical wound was clean and the swelling disappearing. The rise in temperature could not be accounted for, and the ears were examined. Both membranæ tympanorum were found to be normal. Within twenty-four hours the temperature fell to 99.6°, but rose again in the evening. A blood count at this time showed general count, 14,700; polymorphonuclears, 82 per cent; small, 34 per cent; large and transitional, 6 per cent; eosinophiles, 1 per cent.

On January 24th the temperature had remained about 102° all day, with only slight remission. In the evening there was a sudden rise to 103.6°, accompanied by a moderate convulsion and emesis of curdled milk. The urine, which had been negative since admission, now showed a trace of albumin, a few pus cells and hyaline casts, and as the kidney was thought to be the cause of the convulsion, treatment was at once started to relieve this condition. The temperature continued between 103° and 104° for a period of twenty-four hours, when it fell to 102°, only to rise rapidly after another convulsion. The pulse rate was affected, but the respirations were not disturbed.

Again a thorough physical examination was made, which revealed nothing. The wound in the neck was clean, and the wet dressings were discontinued and dry dressings applied. A second leucocyte count was: general, 14,260; polymorphonuclears, 79 per cent; small, 38 per cent; large and transitional, 16 per cent. The temperature continued to run a septic type.

A few erythematous nodules made their appearance on the lower leg and also on the thigh. The heart was weak and rapid; no murmurs were heard. A blood culture taken at this time showed a marked streptococcemia. The ears were again examined, and the drums were normal. There was no mastoid tenderness. There was a thin serous discharge from the cervical wound in the neck.

January 31st. The heart was weak and rapid. Throughout the lungs were scattered moist rales. There was slight involvement of the right elbow joint, with tenderness and swelling.

February 1st. The general picture had become markedly worse. The heart sounds were almost inaudible, the lungs

were filling up with moist rales (a hypostatic congestion). The skin of the lower extremities showed nodular erythema with small macular petechial areas. There was no flush on the body. Both elbows and the metacarpophalangeal joints of the right hand were swollen and slightly discolored. The pupils were irregular, the right dilated, the left side of the face swollen. There was slight meningeal irritation, constant restlessness and moaning. A lumbar puncture was performed and 30 cc. of clear fluid under pressure drawn off. The patient died in a convulsion at 4:20 p. m.

THE AUTOPSY.

The brain was congested, there was an excess of cerebrospinal fluid. There was no meningitis.

The sinuses were thoroughly explored and all found to be free with the exception of the right sigmoid, which contained a septic thrombus that extended from the jugular bulb to and beyond the knee of the sinus.

Both membranæ tympanorum were now incised, and cultures and smears taken from the middle ear.

The mastoid process was removed with the dura intact covering the thrombus.

A dissection along the vein at the site of the abscess revealed a necrotic mass of glands which were removed; the vein was also necrotic and plugged with a thrombus which extended up to the bulb a distance of about one inch; below the vein was collapsed.

The heart muscle was flabby, but there was no evidence of an ulcerated endocarditis. The lungs showed hypostatic congestion. The liver and spleen showed no infarcts. The kidneys were congested and swollen. The joints contained pyemic abscesses. The vein which was removed has been lost and unfortunately does not complete the picture.

The cultures and smears from the middle ears were negative. Smears and cultures from the mastoid were also negative. The sinus and vein showed a streptococcus. The cerebrospinal fluid was negative.

L.

FIBROMA OR FIBROANGIOMA OF THE MIDDLE EAR.

By A. J. Brady, M. D.,

Sydney, Australia.

During an experience of twenty-seven years in a large private and hospital practice as aural surgeon, three cases of peculiar new growths arising in the middle ear have been treated by me. I think it worth while recording my experience of the same, the more so as any attempts to gain information from the experience of others in this condition by reference to standard works on diseases of the ear, did not meet with success. Of the three cases two occurred in women and one in a man—all were adults, ranging from 22 to 27 years of age.

CLINICAL APPEARANCE AND SYMPTOMS.

A smooth firm growth covered with epithelium occupies the external auditory canal, and shows in the meatus. The growth is firm and tough and will not yield to the application of a snare, like an ordinary aural polypus; but the characteristic mark of these growths is the extremely free hemorrhage which takes place when any attempt is made to remove them, or follows even the free application of the probe.

ETIOLOGY.

These growths do not arise, like ordinary aural polypi, as the result of middle ear suppuration. They are probably of similar origin to nasopharyngeal fibroma, and due to developmental irregularities at puberty.

PATHOLOGY.

The growth consists of fibrous tissue with a very large supply of blood vessels. One growth, which was examined by an experienced pathologist, was pronounced to be a sarcoma. I

could not agree with this, as the clinical history and signs were against such. A sarcoma could not exist for several years, during which attempts had been made to remove it, without invading the surrounding tissues. The pathologist's description of the growth, however, was no doubt correct, as he said it was very rich in blood vessels and contained fibrous tissue.

Case 1 occurred in the early years of my practice, and I have not been able to follow up the subsequent history, beyond the knowledge that the growth appeared to be arrested. In this case the growth was removed with the galvanocautery snare, and a few subsequent applications of the galvanocautery were made to the stump—the hemorrhage was not severe.

Case 2 was treated by me seven years ago, and has been recently seen by me. The ear remains perfectly healed, and a firm layer of epithelium occupies the cul-de-sac at the inner end of the auditory canal. The ear is useless for hearing, but this does not trouble the patient, as the other ear is good and the operated one was useless for hearing for years before the operation. The notes in this case concern a young woman, aged 22 years, with a history of ear trouble existing for a number of years. For two years before I saw her she was under the care of a competent aural surgeon, who made several unsuccessful attempts to remove the growth. According to her description, when an attempt was about to be made, a basin had to be ready to catch the blood, which poured out freely. I came to the conclusion that no attempt at removing the growth through the meatus would succeed. I determined to follow the procedure adopted by me in dealing with naso-pharyngeal fibroma, namely, by enucleation (see *Journal of Laryngology*, July, 1906, p. 315). The usual steps of a radical mastoid operation, with cutting down of the postmeatal wall, were followed; then with a fine raspatory an attempt was made to separate the growth from its attachments to the bone—these were deeply situated towards the inner opening of the eustachian tube. The operation was not completed on this occasion, as the bleeding was so severe that I thought it safer to finish it at a later stage. Strips of sterile gauze were packed firmly in the wound, and the bleeding stopped; a few stitches were inserted in the flaps over the packing. Three days afterwards the dressings were removed and the opera-

tion completed by stripping the growth off the bone with a raspatory while it was drawn upon with a tenaculum forceps. The bleeding, although free, was not serious. A month or so later there were indications that the growth was recurring, and several applications of salicylic acid in spirit were made to it without causing it to shrink. Finally chromic acid fused on a platinum point was inserted in the growth. This was followed, almost immediately, by facial paralysis, which did not finally get well for about eight months. After the chromic acid application glycerin of Papain was used as drops in the ear; this seemed to have a good effect in cleaning up the parts, the ear healed and has remained so ever since.

Case 3 was operated on two years ago, a man 27 years of age. I first attempted to remove this growth through the natural channel, under a general anesthetic; but it recurred. At a subsequent operation I turned the ear forward (as in Case 2) and removed the growth with a raspatory. After dressings for over a month, finally the ear healed and has so remained.

LI.

REPORT OF A CASE OF MYXOFIBROMA OF THE NASOPHARYNX.

By J. H. Bryan, M. D.,

Washington, D. C.

The report of this case is interesting from the fact that benign growths of this character in the nasopharynx in children are not very common.

Maggie C., an undeveloped and anemic girl, 12 years of age, came under my observation during March of this year, complaining of occlusion of both sides of the nose, headache, noisy respiration, and at times difficulty in swallowing.

Examination of the nose showed the left side quite free, but no air could be drawn through it into the nasopharynx. The right side was so filled with thick mucous secretion that no satisfactory view of the interior could be obtained. After clearing the nose of mucus I could not get any view of the growth. Examination of the mouth showed a large pinkish growth hanging in and filling the nasopharynx almost completely. The growth extended into the pharynx about one-sixteenth of an inch below the free margin of the soft palate. Owing to the great timidity of the child, it was impossible to remove the growth without the aid of a general anesthetic, so she was placed under ether anesthesia and a wire snare was passed well up into the nasophraynx, the growth seized, and after firm traction withdrawn. The hemorrhage following was quite severe for a few moments, but gradually subsided after firm compression by means of a gauze compress passed well up into the nasopharynx. The growth after removal was found to be pinkish in color, quite firm to the touch, and measured three inches in length. Several days after its removal, on posterior rhinoscopic examination, an excellent view of the nasopharynx was obtained, and there were two points which showed an ecchymotic state, either of which might have

been the seat of attachment; one in the roof of the nasopharynx, which contained some adenoid tissue, and the other in the right posterior nares, just above the posterior extremity of the middle turbinal. The latter was probably the point of attachment.

The following is a report of the microscopic examination made by Dr. J. B. Nichols: The tumor is covered with stratified columnar epithelium, and is made up of myxofibromatous and soft fibromatous tissue, containing massive extravasations of blood and fibrin, small blood vessels are plentiful, gland structures nearly absent. Scattered through the substance of the tumor, in places, in more or less dense collections, are an abundance of polymorphonuclear leucocytes, plasma cells, and pigmented phagocytes.

It is impossible to estimate the frequency with which these neoplasms occur, owing to the fact that many cases are not reported, but judging from the number of reported cases, they cannot be considered to be very common.

Myxofibromas may be considered of nasal origin and generally spring from the upper part of the posterior nares, where the nasal mucous membrane is continuous with that lining the nasopharynx. The former may, therefore, be said to partake of the characteristics of both nasal and nasopharyngeal growths, which may be explained by the fact that they spring from the junction of the two cavities, the lining membrane of the two cavities being quite distinct histologically.

SOCIETY PROCEEDINGS.

NEW YORK ACADEMY OF MEDICINE.

SECTION ON OTOLOGY.

Meeting of March 8, 1912.

A Case of Labyrinthine Fistula Diagnosticated Clinically; Diagnosis Corroborated by Operative Findings; Presentation of Patient.

DR. WENDELL C. PHILLIPS said that interest in the affections of the labyrinth had been very much stimulated during the last three years by the work done in this and foreign countries, and he had been disposed to investigate every case that came into his clinics, either at the Post-Graduate Hospital or at the Manhattan Eye, Ear, and Throat Infirmary, which showed any suggestive symptoms, especially vertigo. During the last fall and winter he had investigated every such case that had come to either of these clinics, and had come across two or three very interesting cases of labyrinthitis, the case presented being one.

Sophie S., 26 years of age; married; housekeeper; came to the clinic on December 26, 1911. Had abscess of the left ear when 13 years of age. Since then had a discharge off and on; sometimes the discharge was purulent, and at other times small foul-smelling masses had come from the ear. There had also been slight headache at times. Three weeks before coming to the clinic she had an attack of violent vertigo and disturbance of equilibrium, accompanied by nausea upon introducing her index finger into her ear. The patient said that she had had milder vertiginous attacks before this.

Upon examination she was found to have spontaneous rotatory nystagmus in both directions in the extreme lateral position of the eyes, more marked toward the diseased (left) side than toward the sound (right) side. The Weber was referred to the sound side, and other tests showed that the hearing was lost on the diseased side. Rotation tests showed both labyrinths functionating equally. Caloric test of the diseased ear gave a positive reaction. The fistula test, by

means of compression of the air in the external auditory canal with a Politzer bag, elicited a marked nystagmus toward the diseased ear; and aspiration of the air, an equally marked nystagmus toward the sound ear.

December 30th, radical mastoid operation. A large cholesteatoma, occupying the attic, aditus, and antrum, was found. There was a large defect in the bony horizontal semicircular canal. Several who witnessed the operation saw this defect. A very small probe was carefully introduced into the fistula, and according to the touch and feel, the probe dropped as though into a cavity. Absolutely no force was used, and there was no sensation of having punctured a membrane. It is generally thought that if one of these fistulæ is found it is better to leave it alone, but this probe apparently fell directly into a cavity, and upon slightly turning it forward, without any force whatsoever, there was a slightly movement of whatever tissue there was in the oval window, and with the least possible force it would have gone through the oval window.

Dr. Phillips said that he laid stress upon this information because of the history of the case. With the caloric test the diseased ear gave a positive reaction; the fistula test elicited a marked nystagmus toward the diseased side, and the aspiration test toward the sound side. There was no record made as to whether the divergence of the eyes was in the opposite direction; the rule is that if the horizontal canal is the site of the fistula, there should be a divergent squint to the opposite side. The radical mastoid operation showed a large cholesteatoma occupying the attic, aditus and antrum, and there was a large defect in the horizontal semicircular canal. The vertigo had continued from the first attack previous to the operation. After the operation the patient had vertigo for several days.

Upon examination on January 18th, eighteen days after the operation, the rotation test showed that the left labyrinth was not functionating, and the caloric test was negative. She has completely recovered and goes about performing her usual duties.

DISCUSSION.

Dr. Berens asked what was the result of the rotation test today.

Dr. Phillips replied that the labyrinth on the one side was

not irritable. There was no hearing on that side. The labyrinth was dead.

Dr. Kerrison asked what had been the condition previous to the operation.

Dr. Phillips replied that the cochlea was not functionating previous to the operation. There was no question but that the vestibule was functionating previous to the operation, but the cochlea was dead.

Dr. Berens said that this seemed to be one of those cases where the external semicircular canal had been walled off by the granulation tissue, and Dr. Phillips was to be congratulated on winning out.

Dr. Dench said that it was a very serious proposition to deal with cases of this kind. Given an absolutely dead labyrinth, with no symptoms aside from nystagmus and vertigo, and on operation finding a localized labyrinthitis, he would not take out the labyrinth. He had done that kind of operation in one case with a fistula in the semicircular canal and a corresponding fistula through the oval window. He made complete drainage, and the child died. On the other hand, he had done a good many operations and found fistulæ in the horizontal semicircular canal, some having nystagmus prior to the operation and others having no special symptoms. If you have a fistula, you may have simply a localized labyrinthitis; other cases show nystagmus, etc., and on operation you do not find any labyrinthine fistulæ. Many of the symptoms which occur in a general labyrinthitis may also occur in a circumscribed labyrinthitis or perilabyrinthitis. It has been his rule, where he found a fistula in a horizontal and semicircular canal, with a normal temperature and no symptoms of beginning meningitis, to do as Dr. Phillips had done in this case, only going a little further and clearing up the localized area in the canal, trusting to the processes of nature to hold the borderline intact, and the patients would not have any further infection. That has often been successful, and without other symptoms he would not clear out the entire labyrinth.

On the other hand, if there are symptoms of general infection, with a temperature of 102° or 103,° you are threatened with beginning meningitis, and the complete labyrinth operation is indicated. In these cases you generally find the acoustic portion of the labyrinth affected.

Dr. Dench also mentioned a case in which a man suddenly became deaf and had an attack of vertigo. He was deaf on the other side, due to a traumatism, and applied for treatment, being out of occupation—for the one ear being deaf and the purulent ear becoming suddenly deaf, he could not hear at all. He had been hearing with his suppurating ear for twelve or fourteen years. A diagnosis of perilabyrinthitis was made. The man now hears as well as before the attack. His vertigo disappeared and he has a perfectly dry ear. Had the labyrinth been opened, the man would have been absolutely deaf. We should go slowly in the extirpation or through and through drainage, unless there are positive indications. He had been too rapid in some cases, and believed that the best results would be conserved by a clearing up of the primary focus of infection, and dealing with it as a localized condition, then watching the patient carefully, and with the first symptoms of beginning infection go in and do a thorough extirpation of the labyrinth.

Dr. Berens said that the cases to which he had referred were cases of intrinsic labyrinthitis, and not perilabyrinthitis.

Dr. Eagleton asked about the symptoms that developed after the operation.

Dr. Phillips replied that as he remembered the case, the patient remained in bed and the vertigo continued for several days with some nystagmus, but no rise of temperature and no other symptoms. She was not able to sit up for six or eight days, and the vertigo gradually subsided. She went home after two weeks, and the ear had been dressed at the clinic ever since.

Dr. Eagleton said that as he understood the case, the patient had had nystagmus both ways before the operation; that it was increased one way and lost the other before operation; she had a vertigo and was put to bed, and was better for a time than before the operation.

Dr. Phillips replied that she was possibly a little better.

Dr. Eagleton replied that Dr. Phillips was certainly to be congratulated upon the outcome of the case. Before so much was known about the labyrinth, these were just the cases that were lost in doing the radical operation. As he understood Dr. Phillips, this girl had an irritable labyrinth, which was probably walled off by granulation tissue. The labyrinth

was destroyed by the operation, and in doing this the natural barriers that were built up were broken down, and such cases were apt to light up a meningitis. During the last few months he had seen a number of these cases giving fistula symptoms. As with Dr. Phillips, he had been in the habit of testing very carefully all cases that go to radical operation, bearing in mind three cases in which he felt he was the cause of the immediate death of the patients. One of these had occurred within the last year. In that instance the labyrinth was functionating, but owing to a too active investigation of the inner wall of the middle ear, a meningitis was lighted up. Since then these cases have been handled with the greatest care. A complete radical operation is done. Where a fistula is found, and these occur especially in the cholesteatoma cases, care is taken not to break down the natural barriers that the granulating tissue has formed. He was strongly of Dr. Dench's opinion that the labyrinth should be left alone if it was functionating. If a labyrinth functionates under any tests, in no way enter it. He had seen cases where by clearing out the cholesteatoma, the labyrinth was thought to be dead, but later it was found to be functionating. If it is not functionating, it is justifiable to remove the labyrinth, but otherwise, if you manipulate, the patient's life is endangered.

In reply to a question, Dr. Eagleton said that the fistula could be enlarged with a cutting curette. Some years since, he had seen Jansen do this work, leaving the membranous labyrinth intact. That sounds impossible, but it is not. By the use of the probe, especially if you go beyond the exposed area, you may light up a meningitis. Many of the fatalities from radical mastoid fall into this group. The natural barriers were broken through and a purulent meningitis lighted up and the patients died.

Dr. Davis said that the case was interesting because of the probability of more than one avenue of entrance of the infection to the labyrinth. The fact that the fistula was situated in the lateral semicircular canal, and that the acoustic function was destroyed, while the static labyrinth was not, indicates this. The infection that destroyed the acoustic labyrinth probably entered by the oval window; if it had entered by the fistulous opening, it would have destroyed the static labyrinth before reaching the cochlea through the vestibule.

In reporting the case the statement was made that when a delicate probe was inserted into the fistula, it seemed to "drop," and that a disturbance or movement of the tissues in the oval window was observed. When we remember the anatomy and relations of the different components of the labyrinth, it is difficult to understand how such an occurrence could come about. If you introduce a probe into the lateral semicircular canal, directing it forward, following the arc of the canal, when you approach the ampulla, the direction is medianly, and not toward the oval window.

Dr. Myles said that the points which had been brought out in the discussion were most important. Following the activity of Jansen, we have been a little bold in the deeper structures of the ear, especially those contiguous to the intracranial structures. Much of this work has been experimental, and it would seem that the value of nature's protective measures had not been sufficiently considered. There now seemed to be more tendency to an appreciation of these factors; it has to come eventually, and the sooner the better. This question had been threshed out very thoroughly in the abdomen, and in cases of appendicitis, where the surgeons have very much changed their ideas, and now look for nature to protect the other parts of the abdomen against the operative field. The doctrine of noninterference in these cases seems to be very discreet; and his experience in past years has led him to believe that his own cases get well largely because of his discretion. At one time he had presented before the Section a complete cochlea and labyrinth removed entirely by nature's processes after the application of some form of caustic placed in the ear, which had destroyed the labyrinth; it had come out intact, and the patient today is perfectly well. He had had several cases where a general curettage around the apparatus has been sufficient to bring about recovery. A few years since he had had a case of tuberculosis of the labyrinth with complete destruction of the inner plate, and by a little slowness in removing parts of the labyrinth and cochlea the patient is now well, whereas if he had been bolder at the time, as seemed indicated, the patient would probably have died. He heartily endorsed Dr. Eagleton's ideas.

Dr. Phillips asked that the courtesy of the Section be extended to Dr. Isadore Friesner, which was done.

DR. FRIESNER expressed his appreciation of the courtesy and said that while he appreciated Dr. Dench's point of view, yet he would like to make one statement in regard to the progress of the case after operation, as, through the kindness of Dr. Phillips, he was able to follow it very carefully. It is a well known fact that the more rapid the progress of a pathologic process—be it suppurative or nonsuppurative—over the nerve endings within the internal ear, the more violent the symptoms. He understood why Dr. Eagleton had asked about the after symptoms, and his own thought might inject another doubt into this "terra incognita" of the inner ear. From the tests we have every reason to suppose that the static labyrinth was functionating normally. Now, nothing can be more apoplectiform in its destruction of the nerve endings than the introduction of a probe into a normal labyrinth. Yet instead of violent after symptoms, the after symptoms here were exceedingly mild. There was no extension of the suppurative process into the internal ear. There was no vomiting, and only moderate vertigo; in short, there were none of the severe manifestations that sudden destruction of a normal labyrinth would lead us to expect. Very grave doubts had entered his mind as to whether this was a normal labyrinth, whether it was functionating normally, as the tests would seem to show. He realized, of course, that it was very unusual to have a dead cochlea with apparently normal semicircular canals, but so far as his knowledge went, that seemed to be the status of the case when the patient presented herself for examination.

DR. PHILLIPS expressed his appreciation of the discussion of the case and said that he had reached the conclusion that if the men who are at the head of large clinics wish to get the best results from the large number of patients, they should not fail to question every case very carefully, especially those of chronic otitis media, to ascertain whether or not they have certain symptoms, and if these are found to investigate them as thoroughly as possible. He has his own assistants follow up these cases most closely, especially if they have headaches or vertigo. One or two cases would have escaped proper attention if this thorough investigation had not been made. The tests are made upon the slightest suspicion, and he would advise everyone to follow that routine.

He had very little doubt now that this case was not func-

tionating, but every test had been applied; there was some little difference of opinion until after the noise producing test, there was no question.

Regarding conservatism, he had made up his mind, in this case, at least, that if nature had intended to throw out any walls of protection it had done so, for there were no meningeal symptoms at all, and that determined him not to open the labyrinth. Nothing could ever convince him that his impressions on dropping the probe through the semicircular canal were wrong. He had never felt anything more plain than that it was an empty cavity. So far as he could determine, the probe dropped down and forward, and he could see the pressure upon the oval window.

Paper: A Critical Review of the Pathology of Deafmutism.

(By Invitation.)

By G. Hudson Makuen, M. D.,

Philadelphia.

DISCUSSION.

Dr. Myles moved that a vote of thanks be extended by the Section to Dr. Makuen for his very valuable and interesting paper, and that discussion be omitted on account of the inadvisability of attempting to gild refined gold.

Dr. Kerrison said that he could not think of a more difficult subject upon which to write a paper, and that the Section was certainly indebted to Dr. Makuen for his presentation of the subject.

Dr. Dench expressed his high appreciation of Dr. Makuen's valuable paper on so difficult and puzzling a subject as deafmutism. It was a subject, however, that should attract more attention than it has up to the present time. Dr. Makuen had brought out the reason why it was so difficult to attack it in a practical way. These patients come under the attention of various observers; in their infancy records are made, and the patients go into the world, and the autopsies, if made, are made by someone who has never seen the patient during life. It is of the utmost importance that there should be a correlation of the clinical and autopsy records, if we are going to

know anything about the subject. Dr. Dench said that he believed much can be and will be done by the proper observation of these patients in various institutions, if the plans already begun are carried out—that is, having institutions in the various states to look after these patients. There the records will be kept, and the patients can be followed up much better than has been possible heretofore. We have in New York state institutions for the instruction of patients of this kind, and in the course of a few years the subject will have had more attention, thanks to the labors of men like Dr. Makuen, and we will then be in position to know more of the actual pathology of the condition.

The conditions which Dr. Makuen had enumerated are, as he said, the result of a result, and it is necessary for us to be able to go into the consideration of the clinical history of the patient throughout the entire course of his life, and to correlate that with the autopsy before we can do anything to improve the condition in cases of a mild character.

Dr. Dench said that he was sorry that Dr. Makuen had not touched upon the instruction which he has given in cases of this character. He stands second to none in the instruction of the deafmute, and there is a very large field open to us in this direction. Neither medicine or surgery help us to improve the condition of these poor unfortunates, but very much can be done by following out the systematic course of instruction which has been adopted by Dr. Makuen, who was the first man in this country to formulate a plan that would bring the subject more clearly before the medical men in this country, and has done it in a very thorough and systematic way.

Two of the largest national societies—the American Rhinological, Laryngological and Otological Society, the American Otological Society, and the Otological Section of the American Medical Association—two years ago appointed a committee to take up this subject. Dr. Makuen, who is a member of two of these societies, drew up a syllabus to be sent to every medical institution in this country. Only in a very small number of the colleges was any attention being given to this subject in the regular course of instruction. Dr. Dench was appointed chairman of the committee which had the matter in charge, and the acknowledgments received from the various medical colleges have been most gratifying. All of them,

almost without exception, have responded very cordially, acknowledging receipt of the syllabus, and agreeing that the subject would receive special attention by the chair of otology of every medical college in the country. This paper, therefore, is very opportune, in bringing this subject before the Section, as Dr. Makuen has brought it before the various institutions devoted to medical instruction in the United States and Canada, As soon as the institutions of learning take up this matter and the young medical men understand that something can really be done for deaf children in order to enable them to become useful members of the community, a stimulus will be given to this subject and the interest in it greatly increased. The Section was certainly indebted to Dr. Makuen for coming and presenting this subject in so masterly a manner.

Dr. Phillips said that during the reading of the paper he had been trying to think of some method by which he could distract the attention of the members from the fact that he knew nothing about the pathology of deafmutism, and he congratulated Dr. Dench on the neat way in which he had evaded the subject, by telling how the deafmute should be cared for after it was determined that he was deaf.

He felt convinced, however, that the best results to arise from the presentation of such a paper would come from an earnest attempt to learn something about this subject. There are many institutions for the instruction of the deaf all over the country. No doubt deaths occur in these institutions, and perhaps something could be done to obtain data in these cases. There are one or two institutions in this city for the instruction of deafmutes, and a public school for them on Twenty-third street, and it is one of the signs of progress along this line that there should be such a school. One of the sad facts in connection with this condition is that if you go through the asylums for idiots you would find a small percentage of deafmutes there who are not idiots, but have been paced there as such. These unfortunates should be instructed to be useful citizens.

The subject deserves more attention than it has received in the past, and committees should be organized throughout the country which should be empowered to secure postmortem reports on the deaths of deafmutes, in order to acquire some

positive facts along this line. He hoped that Dr. Makuen would be able to do some work in this direction, and he believed that it could be done.

DR. MAKUEN, in closing the discussion, said that the most important point in connection with the subject was that mere postmortem examinations of deafmutes, without the clinical histories, are of litt]e value. He had tried to bring out in his paper, also, that without some means of comparing the histology of the normal temporal bone and brain with that of the deafmute, not much can be accomplished. It is exceedingly difficult to get all these data together. Browning says, you know, "Never the time and the place and the loved one." The report of Dr. Bliss shows that there are very few institutions in which such thorough clinical examinations have been made as in the Pennsylvania institution, and yet all this valuable material is going to waste because there is no one to follow up the cases and make the postmortem examinations when the opportunity comes.

NEW YORK ACADEMY OF MEDICINE.

SECTION ON OTOLOGY.

Meeting of April 12, 1912.

A Case of Paracoustic Vertigo and Nystagmus.*

By John R. Page, M. D.,

New York.

DISCUSSION.

Dr. Shambaugh said that it is not easy to determine the probable cause of the symptoms in a case of this sort without making a careful examination. The symptoms are quite unusual. He is inclined to believe that the changes noted in the drum membrane are the result of some previous tubal catarrh which has nothing to do with the peculiar symptoms. One frequently sees cases where the drum membrane is fixed in a retracted position as the result of some previous tubal catarrh, and where functional tests detect no disturbance in the hearing. Dr. Shambaugh thinks that the defect in hearing in this case is due to some disturbance in the labyrinth. The positive Rinne, in the presence of the marked defect in the hearing, in itself should decide this question. He is not able to explain satisfactorily the relation between the peculiar nystagmus and the sounds of certain tuning forks. Perhaps the best that one could say in the way of an explanation of this phenomenon is that the person is a neurasthenic.

Dr. Richards said that he had operated upon a case some six or seven years ago in which the entire labyrinth was removed, and subsequent to the operation the patient was made intensely dizzy when sitting in a room in which a piano was being played—so much so that he had to hold himself in his chair to keep from falling.

In reply to a query as to whether the phenomenon bore any relation to any particular note, Dr. Richards replied that he had not examined into that.

*See page 779.

DR. DUEL said that he could confirm all the records just presented, for he had seen the case with Dr. Page and gone over the tests with him. It had been a most interesting problem. It seemed impossible to attribute this very evident disturbance of the eyes (nystagmus) by certain tones to anything other than a reflex through Deiters' nucleus from a vestibular irritation. Whether this was due to some obstruction of the channels of the labyrinth which normally permitted sound waves to be dissipated without affecting the static labyrinth, he could not say; but it was evident that at a certain pitch the vibrations producing the tone also caused this definite vestibular reaction, always manifested by nystagmus in a certain direction.

DR. FRIDENBERG said that it might be of diagnostic value to determine, in Dr. Page's interesting case, whether there was any difference in the reaction to tones of certain pitch, according as they were conveyed by bone or by aerial conduction alone. This might throw some light on the question of whether the labyrinth or the middle ear was the site of the affection. The change to a reaction to tones of a different pitch indicated that it was a labyrinthine reaction. The ocular motor involvement, as indicated by the fixational nystagmus usually accompanying vertigo, was remarkable and unusual. The occasional transfer of sensations from the organ of hearing to that of sight had been noted, generally in neurotic or very imaginative individuals. In these cases, tones are associated with impressions of color. Thus, the tone of a trumpet makes them see red; that of a flute, blue, and so on. This phenomenon has been described as "colored audition," "photophonism," and so on, and aside from the psychoneurotic feature, has been ascribed to anomalous innervation, or cross connection between the auditory and the visual centers. In the case reported by Dr. Richards, showing the specific action of mechanical stimulation of the trunk of the vestibular nerve in the form of the usual sensori-motor reaction, in spite of the complete removal of the semicircular canals, we have merely a confirmation of the well known fact that each nerve reacts with a special sensation to every form of excitation, no matter how stimulated. Thus mechanical, electric or caloric irritation will cause ringing in the ears, or sensation of light. The end organ, cochlea, retina, labyrinth, serves merely to mediate

socalled "adequate" or "specific" stimulation, as by sound or light. There is an exact analogy in the optic nerve where we can elicit flashes of light after removal of the eyeball, and in the peripheral nerves giving sensation of pain in the toes after a limb has been amputated at the knee.

DR. RICHARDS said that from the case which Dr. Page had presented, he would infer that the lesion was not a middle ear, but a labyrinthine lesion, inasmuch as prior to the myringotomy the patient, as he understood, was made dizzy chiefly by the high notes, and subsequent to the myringotomy was also sensitive to rounds corresponding to the lower notes of the scale, upon the basis that high notes ignore to a greater relative degree than low notes, by obstruction to their passage to the labyrinth, which, if the latter were irritable, would be in a position after myringotomy to be affected by the lower notes.

DR. KERRISON said that he understood there had been a change after the myringotomy was performed—not only were the phenomena of nystagmus and vertigo produced by the low notes, but a cessation of the phenomena by the high notes.

DR. PAGE said that the low tone limit was always normal, and that the myringotomy was done somewhat as an afterthought while the patient was still under the anesthetic given for the lumbar puncture, because of the marked retraction of the drum membrane. The lumbar puncture was done for his vertigo. How much influence the myringotomy had on the lowering of the tones that affected his equilibrium, he could not say, but the upper tones which had previously disturbed him no longer did so. The disturbing tones are not now very low, but they are considerably lower than they were. Before the operation was done the patient had not noted any trouble from his own voice, and now when he speaks in a low tone he is not disturbed, but if he raises his voice on certain words, or laughs—a peculiar high pitched laugh—he is disturbed by it. He did not have that trouble before the operation. Dr. Page said that he could not say whether this was due to air conduction or not, but he is under the impression, because of the effect produced by friction of the hand on the head and ear, that it is through bone conduction that the patient's own voice has the most effect.

Paper: A Discussion of the Theory of the Physiology of the Semi-Circular Canals

(*By Invitation.*)

By George E. Shambaugh, M. D.,

Chicago.

Dr. Shambaugh pointed out that the stimulation of the haircells of the crista ampullaris is the result of a physical reaction, the impaction of an endolymph current against the sides of the cupola. For this reason it should be possible to get much closer to a complete analysis of the reactions leading to the stimulation of this end organ than is the case with other special sense organs. The fact that these reactions in the semicircular canals are in the nature of gross physical reactions, makes it specially important that any attempt to analyze them should be based on a careful anatomic study. Anatomic knowledge, to be of any real assistance, must be acquired by a first-hand study of the preparations themselves.

A number of preparations of the crista ampullaris were demonstrated from lantern slides, and the anatomic reasons were pointed out for the conclusion that the stimulation of the haircells resulting from an endolymph current could last only so long as the endolymph current lasts. It was pointed out that in the Breuer theory the stimulation of the haircells continues for some time after the cessation of the endolymph current, due to changes in the endorgan produced by the endolymph current, whereas in Bárány's theory the reactions resulting from the stimulation of the haircells continue for some time after the peripheral stimulation ceases, due to the expenditure of stored energy in nystagmus centers.

Dr. Shambaugh discussed the reasons, based upon clinical phenomena, for the conclusion that the reactions resulting from the stimulation of the haircells of the crista could continue only so long as the endolymph current lasts, and pointed out that this conclusion has a wide significance in the physiology of the semicircular canals, since it disposes of the necessity for tonus centers to control nystagmus, and places the origin of labyrinth tonus in the haircells of the cristæ.

In discussing labyrinth tonus, the theories of Ewald, Breuer, Sidney Scott, and Bárány, regarding the origin of labyrinth

tonus, were given. Dr. Shambaugh then presented his conclusions regarding the origin of labyrinth tonus, stating that these impulses emanate from the haircells of the cristæ, probably the direct result of an active stimulation of the haircells, brought about by an interaction between the cupola and the hairs of the haircells as the result of intralabyrinth pulsation dependent on the pulsations of the heart. It was pointed out that in each semicircular canal the haircells on the side of the crista receiving the impact of those endolymph currents which direct nystagmus to the same side, are more sensitive to stimulation than are the haircells on the side of the crista receiving the impact of endolymph currents directing nystagmus to the opposite side. For this reason the tonus impulses in each crista from the haircells which, when stimulated, direct nystagmus to the same side, are stronger than those from the haircells which direct nystagmus to the opposite side. This fact explains why the tonus from one labyrinth, if unrestrained by impulses from the opposite side, always produces nystagmus directed to the same side. It explains also why anything which increases the normal tonus from one labyrinth, as, for example, those conditions which increase the intralabyrinth pulsation, will always produce nystagmus directed to the same side.

The various clinical phenomena resulting from disturbance of the normal tonus were discussed, and the explanation offered by this theory of the origin of labyrinth tonus was compared with the explanation of those phenomena offered by the theory of tonus centers.

Dr. Shambaugh expressed the belief that as we learn to know more and more about the reactions in the peripheral mechanism in the labyrinth, that the explanation for most of the phenomena which Bárány has pointed out as objections to the peripheral origin of nystagmus will be found here. As an example of this he called attention to the phenomena of the maximum after nystagmus following ten rotations, and the shorter after nystagmus following five rotations, as well as that following rotations prolonged longer than the ten. These are phenomena that are not accounted for in the Breuer hypothesis, and they are urged by Bárány as objections to the peripheral origin of nystagmus. Bárány asserts that they must find their explanation in some peculiarity in his sup-

posed nystagmus centers. The same thing is urged regarding the phenomenon of after nystagmus following prolonged rotation. Dr. Shambaugh pointed out how all of these phenomena find a satisfactory explanation by the reactions in the peripheral mechanism based upon the theory he has advanced regarding the time relation between the duration of the endolymph current and the resulting nystagmus. These reactions are dependent upon the phenomena of fatigue, and have been described elsewhere.

DISCUSSION.

Dr. Philip D. Kerrison said that Dr. Shambaugh's paper was one of the most instructive and interesting that had been read before the Section in many a day. We are apt to regard papers on so intricate a subject as the physiology of the semicircular canals as theoretical rather than practical. When we have time, however, to analyze Dr. Shambaugh's paper, we shall find in it not only much that is of scientific interest, but also much that is of very practical value. For example, Dr. Shambaugh's theory as to the reasons for the more prolonged nystagmus resulting from an endolymph movement in any particular canal toward its ampulla, as compared with that following a reversed endolymph movement, and his explanation of the fact that after destruction of one labyrinth the duration of nystagmus following an endolymph movement toward the small end of the canal gradually approaches and finally equals that following an endolymph movement towards its ampulla, have a very practical bearing upon certain clinical phenomena sometimes observed. There have, for instance, been reported during the past year several cases in which within one year after removal of one labyrinth the after nystagmus has been the same following rotation in either direction. In one such case, reported before the New York Otological Society, a well-known otologist had drawn a tentative deduction that the function of the labyrinth operated upon was returning. This, of course, was clearly and obviously incorrect. Dr. Shambaugh's paper will make it impossible, however, for anyone in future to arrive at so illogical a conclusion.

A part of Dr. Shambaugh's paper which is of unquestionable value in explaining certain phenomena of vestibular irritation is that in support of his contention that the duration of

nystagmus is in all cases coequal with the duration of the endolymph movement. This explains why we have a nystagmus lasting on an average two minutes after the caloric test, forty-two seconds after the rotation test, and the momentary nystagmus observed in Ewald's experiments. In Ewald's experiment the endolymph movement lasts only during actual compression, any continued endolymph movement being prevented by the mechanical occlusion of the canal at some point. That the resulting nystagmus lasts only while the endolymph is actually subjected to pressure is in exact accord with Dr. Shambaugh's theory.

DR. DUEL said that the hour was late for continuation of the discussion, and that it might be well to ascertain the wishes of the Section. (Moved and carried that the discussion be continued.)

Dr. Duel then congratulated Dr. Shambaugh on his able presentation of this most interesting and important subject. It was one over which he had "dreamed" for years, and his heart and mind were full of it, yet he approached the discussion with great diffidence, owing to the fact that if one differed from any of the points which Dr. Shambaugh had brought out, it would take nearly as long to present the dissenting idea as had been required by the author to present his views. He would, therefore, content himself with saying that while his own knowledge of the subject had not resulted from such an arduous anatomic study as Dr. Shambaugh had made, theoretically he had evolved some ideas which to a certain extent coincided with and yet in others differed from those presented by the author.

The very constant phenomena which resulted from stimulation of the vestibular apparatus by heat or cold, turning with the head in different position, galvanic currents, and pathologic conditions, in his opinion, might lead to a theory of the anatomic arrangement producing them, almost as accurate as a minute study of the anatomy, although, of course, not as convincing. Ideas evolved from such different points of view were doubly interesting when they agreed. Dr. Shambaugh had shown some very plausible anatomic reasons which made the theories of Breuer and Bárány seem untenable. These objections had been supported by observations of phenomena which had long since impelled him to abandon their theories for a more plausible one very similar to that of the author.

Dr. Duel mentioned additional well known phenomena in support of Dr. Shambaugh's idea. In regard to the question of tonus, the time was too short to enter into any intelligent discussion of it. In the main he agreed with the position taken by the author, particularly with the refutation he had made of the dogmatic statements of Newmann and Bárány. The question of the centers from which these tonus influences come was a long and difficult one, and, of course, a highly theoretic one.

In the same way it seemed to him impossible that Bárány's theory that it was tipped over until it upset the cap which set off the magazine, producing certain phenomena of nystagmus until that was exhausted, could be accounted for by the facts.

Dr. Fridenberg said that the Section was to be congratulated on having Dr. Shambaugh give them the results of his minute and painstaking investigations. These had, above all, given a definite anatomic basis for hypotheses and added greatly to the logical quality of the theories which had been advanced by Dr. Shambaugh. This had been the case for the speaker, especially in regard to two points. One was the histologic study showing that the cupola cannot move as a whole, and that the reaction to endolymph current takes place mainly on the side of the crista ampullaris. The modification of theories of adequate stimulation by which endolymph stress had been substituted for actual endolymph motion in currents, was corroborated by these findings, and Dr. Shambaugh's theory of instantaneous excitation and synchronous character of stimulation and labyrinthine reaction was made logical and easy to understand. Another interesting point brought out by Dr. Shambaugh was the part played by the vestibule itself, acting as a larger segment continuous with the narrow canals, and thus supplying a quantity of fluid quite sufficient to give bulk and force to mechanical stimulation of the ampulla. This was a novel and logical answer to the objections that the canals were of too fine a bore to allow any current of endolymph. The speaker considered this point of great practical importance, as the mechanism not only gave a sufficient volume of fluid, but this volume was actually intensified in its action by being carried in and through a narrow canal, as through a funnel, to the exact point at the side of the crista where it would act most effectively. It was, as it were,

focused on the crista by this means. The logical working out of the effect of endolymph stress and of the effect of endotic pulsations and currents for the normal stimulation of the "quiescent" labyrinth were of the greatest interest, and could not fail to advance our knowledge of labyrinthine reactions in disease. There was but one point which had been doubtful in the speaker's mind. This was the gradual development of compensatory tonus, after destruction of one labyrinth, in those crista cells of the opposite labyrinth which ordinarily had only a minor function. This theory was very enticing and appeared to answer the hypothetic requirements perfectly. The missing link was the modus of stimulation of these cells. For compensatory hypertrophy one would have to have increased function, depending on frequent irritation or stimulation, and as these contralateral cells reacted only to rotation in one direction, this should imply that the patient would have to be rotated repeatedly in one direction to cause these cells to hypertrophy. He had no doubt that Dr. Shambaugh would explain the mechanism and clear up this doubtful point. Attention might be called, in closing, to another theory of the labyrinth, in which all the reactions are ascribed, not to irritation of central organs, but to an inhibitive control. This inhibition is supposed to regulate, check and measure the motor impulses constantly flowing from the cerebellum subconsciously to the skeletal muscles, in answer to the multitude of sensations received from limbs, viscera, integument, and also from eye and ear. It is this regulation of motor impulses which results in tonus and in coordination. The theory has been elaborated and is very captivating, as it explains many points which are not easily understood on the basis of the Breuer-Mach hypothesis. The reaction just mentioned of equalization with gradual destruction of a labyrinth is explained by this theory, on the basis of a gradual loss of the inhibiting control and a corresponding adaptation in the central organ, i. e., cerebellum, whereas after acute destruction of the labyrinth the sudden loss of control results in a marked reaction. It has even been claimed that all afferent nerve impulses act indirectly to produce tonus, and that sensations of touch, sight, muscular innervation, pressure, and so on, are actively concerned.

Dr. Richards said that the supposition of Scott—as men-

tioned by Dr. Shambaugh—in attributing tonus impulses to the pulsations of the carotid in its canal (these being transmitted regularly to the labyrinth) was altogether a theoretic supposition and one which could be shown to be incorrect. For the carotid artery in its canal can be shown upon exposure to possess practically no pulsation. In fact the carotid canal, as he had pointed out several years ago, performs the double function of shielding the brain from excessive pulse impact and of converting the flow within it from a remittent into a more continuous stream. In reference to Ewald's experiments, he said that the labyrinth is an exceptionally difficult organ to investigate experimentally, as it is filled with a fluid medium which is continuous. A disturbance at one part (whether it be either injury or pressure) is transmitted throughout its entire extent, and the resulting phenomena which follow may be due to the irritation or disturbance of distant parts, as well as to the local point of experiment.

Another point of interest on which clinical observation throws some light is the importance of the entire canal system as a necessary factor either in maintaining muscle tonus or equilibrium. We see cases in which, after one labyrinth is destroyed, nystagmus and vertigo quickly disappear; now, if compensation is established by the opposite side, we would scarcely expect to see it occur in so short a time as we do sometimes see it. I have operated on cases where immediately following the destruction of one labyrinth there has been a total cessation of the nystagmus and vertigo; certainly, there has not been time for the opposite ear cells to take on such additional power, any more than if we remove one eye the other eye would suddenly increase its vision. My own belief is that the function of the semicircular canal is not nearly so important as it is generally supposed to be. I saw a case recently in which the patient gave no response to the caloric or whirling tests, but had become totally readjusted.

Dr. Shambaugh (closing discussion), replying to Dr. Richards, stated that it is quite true that the close anatomic relation between the several parts of the internal ear precludes the possibility of an opening in one part with escape of fluid taking place, without all parts being affected at the same time. In fistula cases referred to, and in the Ewald experiment, the opening is only through the bony capsule and not through the

endosteum lining the cavities of the labyrinth. Here there is no escape of labyrinth fluid. Dr. Fridenberg asks how it is possible for the haircells on the less sensitive side of a crista to take on a compensating increase in sensitiveness so as to equal the cells on the more sensitive side after the destruction of one labyrinth, since there is no increased stimulation of these cells. Dr. Shambaugh does not think that this offers a fundamental objection to the theory that such a compensating increase in function does take place. Similar phenomena are observed in other organs of the body, where a compensatory increase in function takes place without our being able to say just why it does develop. Dr. Fridenberg also questioned how it is possible for a compensatory increase in the activity of these haircells to take place so rapidly as to account for the rapid disappearance of the disturbance of equilibrium in some of the cases of unilateral destruction of the labyrinth. Dr. Shambaugh pointed out that it has only been in cases of long standing destruction of one labyrinth where the rotation tests have indicated that such a compensatory increase in the activity of the haircells had taken place. The evidence of such a condition is the fact that in applying the rotation test the after nystagmus to the affected side, which results from a stimulation of the cells on the normally less sensitive side of the crista, is as strong as the after nystagmus to the normal side, which is dependent on the stimulation of the haircells on the normally more sensitive side of the crista.

NEW YORK ACADEMY OF MEDICINE.

SECTION ON OTOLOGY.

Meeting of May 10, 1912.

Report of Case of Primary Lateral Sinus Thrombosis With Normal Mastoid and Tympanum.*

By H. L. Lynah, M. D.,

New York.

DISCUSSION.

Dr. Lynah, in reply to a query from Dr. Kerrison, said that the clot extended from the bend of the lateral sinus to the jugular bulb.

Dr. Kerrison said that there had evidently been no mistake as to the presence of a septic clot extending well above and below the jugular bulb. The most interesting question was as to where the infection originated. In spite of the absence of evidences of a suppurative process, past or present, in the tympanum or mastoid process, Dr. Kerrison believed that the infection had most probably originated in such a lesion, which had subsided after giving rise to a focus of infection within the sinus.

Dr. Heller said that a peculiar point was the small leucocytosis. One would expect more than 14,000 white blood cells. This case recalled to mind one reported by Dr. Hayes several months before, with a history of a primary mastoiditis in which the leucocytosis was less than 10,000. Where children are so very sick and have a small leucocytosis, the prognosis is very much worse than where there is a leucocytosis of 30,000; the resistance is lowered and the body is not throwing off enough white blood cells to protect it from the infection.

Dr. Kerrison said that Dr. Heller's point in regard to leucocytosis was very interesting. Dr. Kerrison referred to a most interesting paper on ligation of the jugular vein by Dr. Crockett, of Boston, in which the writer stated his belief that a rather low leucocyte count in sinus thrombosis was a good differential point between that lesion and purulent leptomeningitis, in which the leucocytosis was usually comparatively high.

*See page 784.

DR. PAGE said that about two and one-half years ago he had reported a case in which the lateral sinus was apparently normal inside and out, but there was a clot in the bulb. This case had practically a leucopenia, so much so that typhoid was suspected, and a jugular resection was delayed for three or four days on account of it. Several blood counts were taken, some being between 4,000 and 5,000, and none were higher than 10,000 until after the vein was resected. The patient had a metastasis in the left wrist and a paralysis of the right external rectus. Dr. Page said that he had not heard in Dr. Lynah's paper what the onset of the trouble was, but understood that the patient had been operated upon first for cervical abscess. He had seen many instances in babies in which the discharge from the ears had been overlooked by the parents, but in which there was a scant discharge with enlarged glands below the mastoid, and he had found that when this enlargement persisted, the mastoid was usually involved. Instead of putting such children under irrigation, he had advised exploration of the mastoid as the quickest way of clearing up the trouble, and in every such case that he had operated he had found the mastoid involved. It is difficult to decide in this case between a primary bulb thrombosis and a direct extension from the glands to the vein.

DR. BALLIN said that an important point in the case reported by Dr. Lynah was the blood culture. It was found to be positive. Of course this subject had often been discussed before. Here was a case with a thrombosis and a positive blood culture. In most of these cases at Mt. Sinai Hospital it was found that where there was a suspected sinus thrombosis and a positive blood culture, even without a marked rising and falling temperature, one could be pretty sure that there was a sinus thrombosis. Most of these cases of infection take place through the middle ear. This may have been one of those cases in which the diphtheritic otitis media had been overlooked and in which the infection nevertheless took place through the middle ear, working its way into the jugular bulb and later developing into a sinus thrombosis.

DR. LYNAH said that the child was doing well and the temperature had fallen to normal on the 22d, five days after admission.

The rise in temperature the following day could not be

accounted for, and was therefore attributed to the cervical abscess. He thought that this condition of sinus thrombosis may have occurred before in scarlet fever and diphtheria following suppurative cervical adenitis, and he had been on the lookout for others and had carefully followed all cases of cervical abscess, but had been unable to find any evidence of a thrombus at autopsy.

It was interesting, however, to note that in scarlet fever and diphtheria complicated with cervical abscess, thrombosis of the internal jugular vein or the sigmoid sinus may occur independent of any middle ear or mastoid involvement. He believed that the infection followed through the jugular vein and thence to the sinus, for the jugular was found to be blocked with a collapsed vein below. Whether the pressure of the cervical cellulitis primarily caused a blocking of the vein and the infection occurred later, he was unable to say. It was an interesting case from the standpoint of both the otologist and the pediatrician.

Paper: Syphilis of the Inner Ear.*

By Otto Glogau, M. D.,

New York.

DISCUSSION.

Dr. Kerrison said that he regarded Dr. Glogau's paper as a valuable contribution to the subject. If we will look into the chapters on syphilis of the inner ear in most textbooks, the meager treatment of the subject will show the large amount of work which had gone into the production of Dr. Glogau's paper.

Dr. Page said that Dr. Glogau had spoken of the upper tone limits and the high forks, and wished to know whether he used the Galton whistle or relied entirely on the forks.

Dr. Heller said that he had not had many cases of secondary syphilis of the inner ear, but this fact certainly had come to his mind more than once, and in all cases of socalled catarrhal otitis media chronica he had made it a part of his routine practice to give potassium iodid. He admitted that this was purely empirical, and that he had no reason to suspect syphilis in all of these cases, but he did find that on giv-

*See page 703.

ing them five grains of potassium iodid, perhaps combined with potassium bromid, he got good results, and better than from other forms of treatment. It may have been that some of these cases had had syphilis. He recalled two cases very distinctly, one of them resembling a case reported by Dr. Yates a year ago, where the tinnitus was so loud that one could hear it; in this instance he gave the patient potassium iodid and bromid, and the trouble disappeared. Whether that was merely a coincidence, or whatever was the cause, he now gives the potassium in these cases without waiting.

Dr. A. J. Herzog said he had two cases of tertiary syphilis of the auditory nerve, in which the hearing was acutely lost. The only drug giving any result were deep gluteal injections of salicylate of mercury in albolene suspension. The result was a slight improvement only.

Dr. Glogau, in closing the discussion, said that at the dispensary he uses high tuning forks of 20 to 48 vibrations; they are practically high enough to determine the upper limits. The other two tuning forks which he uses are the low one, the 32, of the Vienna school, and a middle one of 435 vibrations; the latter is used for Weber, Rinne and Schwabach tests. He first examines with the low fork, and then brings the middle fork to vibrate. Putting it on the forehead, he determines Weber's test; holding it before the ear and putting it on the mastoid, he finds out if Rinne is positive. He touches somewhat the tuning fork so as not to let it stop of itself, and finds out whether the bone conduction is shortened or not. While thus testing for Schwabach's test, he withdraws at intervals the fork from the mastoid, otherwise Gradenigo's symptoms of nerve exhaustibility occurs. Thus combining all three tests together, much time is saved in dispensary work.

Dr. Heller said that he gave potassium iodid, and Dr. Glogau suggested that in these cases he should try sajodin, which he himself had found very useful. It was his experience that 80 per cent of the cases of otosclerosis have syphilis as an underlying condition, either acquired or inherited, perhaps one or two generations before. He had not seen the case mentioned by Dr. Keller where the bystanders could hear the tinnitus. He did not know whether the doctor had treated his case with antisyphilitic treatment or not, but he knew that spasms of the eustachian tube, the only condition

where cracking noises can be heard by bystanders, are not easily amenable to treatment.

Dr. Kerrison had mentioned salvarsan in the treatment of lesions of the inner ear. Dr. Beck and others in Vienna described many cases where after injections of salvarsan the acoustic nerve became perfectly deaf, or the static apparatus became affected where before there were no symptoms. On the other hand, they described cases where impairment of hearing caused by luetic infection, or other static condition, disappeared after salvarsan. In one case there was only a lesion of the static apparatus of the inner ear, and after injection of salvarsan it disappeared.

The preparation of this paper had entailed a great deal of work, as he had to read through the literature of the last hundred years. In all the American textbooks he found only a few lines mentioning syphilitic changes of the inner ear. In Politzer's book there are four pages dealing with this subject. The only book that is really valuable in relation to this subject is Gerber's comprehensive work on syphilis of the nose, throat and ear. He hoped that this would soon be published in English.

ABSTRACTS FROM CURRENT LITERATURE.

I.—EAR.

The Determination of Caloric Nystagmus by the Use of Cold Air.

KALLMAN, Berlin (*Passow and Schaefer's Beitrage zur Anatomie des Ohres,* August, 1911, Band 5, Heft 2). In this article the author gives a complete historical review of the subject and appends an up to date bibliography of over 200 references. His results are not essentially different from those obtained with cold water. The main advantage of the method is the use of air when a perforation is present. *Horn.*

The Determination of the Relation Between Air and Bone Conduction by Means of the Monochord and the Tuning Forks.

KALLMAN, Berlin (*Passow and Schaefer's Beitrage zur Anatomie des Ohres,* August, 1911, Band 5, Heft 2). In a very carefully worked out series of experiments on forty-four individuals, the author has shown the great value of the monochord as an addition to our otologic diagnostic instrumentarium, and also brought out some new points. The subject is a complicated one, and for more detailed information one should consult the original. In this article he proves that the highest monochord tones are heard better by bone conduction than by air, and that the greatest difference is found in patients with an internal ear condition (nervous deafness). In the normal individual and in diseases of the conducting apparatus the relation remains the same.

In the higher tones the amplitude can be very much exaggerated by fastening to the end of the monochord a metal plate 4½ cm. square. This makes a difference in the perception of as much as three or four centimeters in the length of the wire.

The author having proved that the higher tones of the monochord are better heard by bone conduction, the natural question presented itself for solution whether the middle and lower tones were also better heard by bone than by air conduction. Struycken maintains that the improved hearing by bone conduction is independent of the height of the tone, but is influenced by the amplitude. The present experiments show

that the tones up to c^2 are better heard through the air than the bone. From this point upwards the tones are better heard by bone conduction.

In a footnote he makes this interesting statement: "The diagnosis of ankylosis of the stapes is made when with a normal drum the Rinne up to c^2 is negative, and the deeper tones are shortened more than the upper, the upper tone limit remaining normal. *Horn.*

Contribution Concerning the Etiology of Tinnitus Aurium.

Otto Mayer (*Monats. f. Ohren. u. Laryngo-Rhinologie*, Vol. XLVI, 1912, p. 201), after careful consideration of the various theories concerning the cause of tinnitus of otitic origin, has carried out a series of examinations of eighty temporal bones, and especially the findings in a case of leucocythemia. He concludes that in the large majority of cases of disease of the inner ear the tinnitus is due to an autoauscultation of the increased circulation. In the case reported he believes that tinnitus of the left ear was produced in this manner. In the right ear the perilymphatic spaces of the cochlea were filled with connective tissue and bone and there was a slight degeneration of ganglion cells and nerve fibers in the lower part of the cochlea, also the leukemic exudate was found in the sacculus and perilymphatic spaces of the ampullæ. Also there was marked dilatation of the blood vessels in the labyrinth and middle ear. He calls attention to the fact that one must be very careful in making his examination to exclude this vascular congestion before accepting the view that a slight degenerative change in the periphereal neurons of the acousticus is responsible for the tinnitus. *Wood.*

Concerning the Ligation of the Juguluar Vein in Cases of Thrombosis of the Lateral Sinus Following Acute Purulent Otitis Media.

Schneider (*Archiv. für Ohrenheilkunde*, Vol. LXXXIX, 1912, p. 75) in case of lateral sinus involvement makes a plea to limit ligation of the internal jugular vein to those cases in which the jugular itself is already thrombosed. His usual method of operating in cases of sinus thrombosis is, after having laid the sinus walls sufficiently bare, to determine by hypodermic puncture the consistency of the interior of the sinus, and in cases where there is a thrombus to remove the external

wall of the sinus and any organized clot that is not too firmly attached. He avoids, however, excessive manipulation and especially the use of a sharp curette. He says that the septic thrombus is almost always cut off from the rest of the blood current by a healthy clot, and all that is necessary is to give the infected portion of the thrombus a sufficiently large exit to insure drainage. He reports thirteen cases of acute thrombosis with only one death, that is, recovery in 92.3 per cent; also ten cases in which there was a suspicion of sinus thrombosis. Of these, nine recovered and one died. He says that these results are better than any so far reported, even where there has been ligation of the jugular. He strongly advises early operations and the avoidance of unnecessary manipulation of the thrombus. *Wood.*

New Method of Mobilizing the Drum Membrane.

MARTIN SUGAR (*Monats. f. Ohren. u. Laryngo-Rhinologie*, Vol. XLVI, 1912, p. 226), believing that were it possible to forcibly draw the drum membrane outward, marked benefit could be obtained in cases of middle ear adhesions, finally developed a technic whereby he could fasten a pledget of absorbent cotton to the surface of the drum without injury to the membrane. He used for this purpose various adhesive materials, finally developing the following formula:

Mastosch	2.5
Dammar	2.5
Resina sandaraca	10.0
Absolute alcohol	5.0
Ether	20.5

Under careful illumination a drop of this solution is placed on the posterior inferior quadrant of the intact drum. A carefully pointed cotton tampon is then dipped in the same material and slightly pushed in place against the drop on the drum. It remains for twenty-four hours, at the end of which time it is firmly fastened. Traction can then easily be made with a pair of forceps applied to the tampon. At each sitting three to four dozen tractions can be made, drawing the drum well outward each time. The cotton becomes loosened of itself in a number of days, and it may be necessary to fasten on a new pledget. This can, however, be done without any damage to the drum. *Wood.*

Concerning Serous Labyrinthitis.

E. RUTTIN (*Monats. f. Ohren. u. Laryngo-Rhinologie,* Vol. XLVI, 1912, p. 233) reports an interesting case of a boy ten years old, who for two days had had headache and pain in the right ear. For twenty-four hours with increase of the pain in the head and earache there developed extreme dizziness and a distinct sense of turning, and he had vomited once. The left ear was normal. The drum of the right ear was red, swollen and macerated, and there was a scar in the posterior portion. Absolute deafness for both speech and tuning fork; Weber, left; Rinne, negative; Schwabach, shortened. There was spontaneous nystagmus to the left. The caloric reaction was absent. No fistula signs. The mastoid process was sensitive over the attic, antrum and region of the sinus, and the jugulars were free. The cervical spine was sensitive to pressure, but active and passive movement possible. All reflexes were increased, but there was no Kernig, no ataxia, and no marked disturbance of the sensations. Température, 37.4° C.; pulse, 104; eye ground normal; lumbar puncture clear, but pressure increased. An exploratory operation was undertaken. The sinus was found to be normal; the mastoid process pneumatic and free, and only in the antrum were a few granulations found, so that the case was probably one of acute otitis. There were no changes in the wall of the labyrinth. At this stage of the operation the case was supposed to be one of tuberculous meningitis, and the operation was stopped. Nystagmus disappeared in two days and the patient felt well. On the sixth day the patient was able to hear conversation; Weber, right, middle fork well heard; Rinne, negative; no spontaneous nystagmus; no fistula symptoms, and caloric reaction was active. There was, however, at this time a slight evidence of beginning facial paresis, which on the next day was quite marked. This case was evidently one of serous labyrinthitis with temporary loss of the function of the labyrinth. *Wood.*

Experimental Research Concerning Galvanic Nystagmus.

MARX (*Archiv. für Ohrenheilkunde,* Vol. LXXXVIII, Heft 1 and 2, p. 2) carried out a series of experiments in guinea pigs by blocking the semicircular canals, or by extensive destruction of the same on one side, and in other cases on both

sides. He found that after complete destruction of the labyrinth it was possible with the galvanic current to produce a normal reaction. Early the reaction was more easily produced on the side of the destroyed labyrinth than on the attacked side, but later a stronger current was necessary to produce the reaction, although the changes in the quality of the reaction did not take place. These results were supported by clinical observations, although they are opposed to the view of Brunning. As a result of this research Marx came to the conclusions: First, that the presence of normal functionating specialized epithelium was not necessary for the production of the galvanic nystagmus; second, that the galvanic test is not a conclusive proof of the condition of the labyrinth. *Wood.*

Experimental Research Concerning Acute Suppuration of the Middle Ear.

HAYMANN (*Archiv. für Ohrenheilkunde,* Vol. LXXXVIII, Heft 1 and 2, p. 2) carried out seventy-three experiments on forty-four guinea pigs. He used as the infecting material staphylococci, streptococci, streptococci mucosa, diplococci, diphtheria bacilli, pyocyaneus bacilli, and, in a few cases, a number of other bacteria.

He found that the inflammatory process progressed unevenly so that in even severe cases some of the mucous membrane remained intact or almost entirely ualtered. The drum membrane in comparatively similar inflammatory changes of the middle ear showed entirely different conditions. Involvement of the ossicles in cases where the mucous membrane covering was diseased frequently showed new formation of bone so that the ossicles became fixed to the neighboring bony walls by newly formed osseous adhesion. These bony changes were the most frequently observed where the streptococus mucosa was the infecting organism. In a number of cases the labyrinth was involved, the infection taking place sometimes through the round window, and sometimes through both. The labyrinth showed different degrees of involvement, varying from a slight early infection to complete filling up of the inside of the labyrinth with bone and connective tissue. In some cases the meninges became infected from the labyrinth, either through the internal auditory meatus or through one of the aqueducts. The aqueductus cochleæ frequently was found to

be free in cases where there was severe suppurative changes in the labyrinth, and this was probably caused by early closure of the aqueduct. *Wood.*

A Case of Otogenous Brain Abscess—Operation—Recovery.

HAMMERSCHLAG (*Wiener medicinische Wochenschrift*, No. 12, 1912). The following case presents some unusually interesting features: The patient, a woman, aged 57 years, had had diabetes for fifteen years. In October, 1911, she developed a right sided acute otitis media which ruptured spontaneously. In spite of the resulting profuse discharge from the ear, she did not improve, the severe pain continuing and a slight swelling developing in the region of the mastoid process. A few days later some slight cerebral disturbances appeared, and at that time the author was first called to see the case. He found the patient confused mentally, speaking in an irrational manner. A symptom was noticed at this time which persisted up to the time of operation. It consisted of convulsions, very much like Jacksonian seizures, coming on about every fifteen minutes. During the convulsive attacks the head was invariably turned sharply to the left and kept in this position for a half minute. The eyes were also turned to the left, and a marked horizontal nystagmus to the left was always noticed.

The patient was sent to a hospital in Vienna, and was observed there by the writer for a number of days before operation; the urine at this time contained 7.1 per cent sugar, and the convulsive seizures occurred every five minutes, with movements of the left hand, the symptoms pointing to a lesion of the right hemisphere. Otherwise the examination of the nervous system was negative, as was also examination of the eyes.

Two days before operation a symptom developed which neither at the time nor later at the operation, could be explained. Occasionally the head and eyes turned to the right with a nystagmus to the right during the attacks. Complete cortical blindness came on at this time. The author believed that all the symptoms could best be explained by an abscess in the temporal lobe, although Jacksonian attacks are rare in abscesses in this part of the brain. Hammerschlag has collected 195 cases of abscess of the temporal lobe from the lit-

erature, and this symptom was present in only six. Redlich, Epstein and Stauder have reported cases of diabetes in which similar brain symptoms were present.

At the operation the entire mastoid process was found softened, some of the cells containing pus. No defect could be found anywhere in the tegmen antri.

The temporal lobe was then exposed and the dura was found to be tense, with no pulsation. Far anterior, corresponding to the position of the aditus ad antrum, a fibrinous deposit was found on the dura. The brain was entered at this point and at the depth of 1 cm. pus was found in considerable amount.

The further course of the case was favorable. On the following day the patient was much more rational. The movements of the head and eyes to the right ceased permanently, and the patient finally left the hospital entirely well.

Theisen.

Further Studies on Otosclerosis.

G. FERRERI (From the Acts of the 14th Italian Congress of Oto-Rhino-Laryngology, Rome, October 26-28, 1911) concludes from a series of observations that otosclerosis is a disease of long duration, secondary to an hereditary condition or a latent autointoxication of the skeleton, the result of rickets and osteomalacia. In rickety children affected with deafness or diminution of hearing, the vaccination on the mastoid region (according to Wright's method) seems to have given the author encouraging results.

The method of Wright consists of a vaccine prepared from cultures of diplococci isolated from the blood of individuals who are affected with osteolmalacia and rickets.

Lucchetti.

Studies and Suggestions in Acumetry.

GRADENIGO AND STEFANINI (From the Acts of the 14th Italian Congress of Oto-Rhino-Laryngology, Rome, October 26-28, 1911) have devised a new and practical acumeter with tuning forks, which is supposed to register the duration and, what is more important, the intensity of the sound, which latter quality has been wanting in many of the instruments used in ear tests.

Gradenigo terms the vocal index the difference between the auditory distance of the whispered and spoken voice v-V. He

uses this with reference to the vowels in diseases of the ear, and he has reached these practical conclusions that

First, in diseases of the internal ear the vowel *i* is very much reduced.

Second, in diseases of the middle ear the *u* is the most reduced.

Third, in the mixed forms (middle and internal) the ones most distinctly heard are *a, o,* and *e*; the least *u* and *i*.

Gradenigo uses the word auditus the same as the oculists use the word visus, and the following formula to express the same:

$$\text{Index Vocalis} \begin{cases} \frac{\text{V}}{\text{V}.=}. \end{cases}$$ *Lucchetti.*

II.—NOSE.

Visual Disturbances Cured or Relieved by Nasal Operation.

E. Baumgarten (*Archiv. für Laryngologie und Rhinologie,* Bd. 26, Heft 1). Conditions reported of amblyopia from orbicular spasm, retrobulbar neuritis, incipient neuritis, acute papillitis, and atrophy of the optic nerve. In these cases there were found sinus infections and various disturbances of the ethmoidal and sphenoidal cells. The rhinologic treatment of these various disturbances were followed by brilliant results. *Goodale.*

Papilloma of the Nose.

W. Brock (*Archiv für Laryngologie und Rhinologie,* Bd. 26, Heft 1) contributes a case to those already known, about forty in all, characterized by cauliflower-like growths, continually recurring after removal, and showing histologically long slender branching papillæ, covered with cylindrical epithelium, situated upon a very loose, delicate, edematous connecting tissue, containing numerous blood vessels. *Goodale.*

A Contribution to Serodiagnosis of Ozena.

Caldera and Gaggia (*Archiv. für Laryngologie und Rhinologie,* Bd. 26, Heft 1) from their observations conclude that ozena has probably no specific cause, although the organisms present in the crusts contribute to the fetor. The general condition of the organism, the local condition of the tissues, and the action of bacteria produces in association the pathologic condition known as ozena. *Goodale.*

On the Relation Between the Maxillary Sinus and the Lacrimal Canal.

J. FEIN (*Archiv. für Laryngologie und Rhinologie,* Bd. 26, Heft 1). The anatomic relationships show the possibility of a mutual extension of infections from one organ to the other, and also the danger of injuring one organ by operative procedure on the other.

Dacryocystitis most frequently finds its cause in suppuration of this sinus. Exploratory puncture should be undertaken wherever doubt exists. Pressure on the duct on the part of the maxillary sinus is rare, but may occur from polypi or dental cysts, and especially in ulcerative processes in the bone.

Goodale.

Ethmoiditis Purulenta Exulcerans with Atrophic Rhinitis.

RUNDSTROM (*Archiv für Laryngologie und Rhinologie,* Bd. 26, Heft 1) reviews the literature and reports cases in which affections of the accessory sinuses were present, not only in atypical forms of ozena, the socalled Grünwald form, but also in typical cases of the Fränkel type. With the healing or improvement of the sinus suppuration a secession of the clinical symptoms of ozena appeared. The secretion of the so-called Fränkel form of ozena dries and produces crusts in the ethmoid cells, although one cannot see any secretion flow from them. Consequently, the diagnosis of sinus involvement cannot be established unless the sinus is exposed and opened by operative procedures. A definite clinical difference exists between a fetid and a nonfetid form, precisely as in the case of atrophic rhinitis; and a definite pathologic anatomic difference exists between these two types, namely, that in the fetid form an ulcerative process occurs in the sinuses, with discoloration of the bony tissue, while in the nonfetid form, the mucous membrane of the sinuses, although chronically inflamed, is intact in its continuity; or if the mucous membrane is destroyed, the bony tissue is still white, smooth and shining. The carious process in these bones cannot be demonstrated by probing, because the desiccated secretion adheres firmly to the carious bone, like the crusts in the nasal passage, as a result of which it is impossible to feel the exposed bone with the probe. Atrophy of the turbinates and abnormal width of the nasal passages are not one and the same thing; but wide nasal passages may be present long before any atrophy, either of the

turbinate bone or of the mucous membrane, and hypertrophy of the mucous membrane may even be present.

Ozena is consequently a clinical picture which arises through a chronic inflammatory process in the mucous membrane covering the ethmoid cells, running a course, usually in the form of a purulent catarrh, and leading to a retention of secretion in the cells in consequence of closure of the channels of exit. This retention of secretion occasions in early life, when the tissue is still soft and yielding, an ectasia and enlargement of the ethmoid labyrinth and consequently widening of the nasal passages, with an alteration of the exterior formation of the nose. Through the inflammatory process, and the increased pressure on the accesory sinuses, the mucous membrane is destroyed, whereupon the process passes over to the bony tissue. In consequence of the increased width of the nasal passages, and the richness of the secretion in formed elements, it undergoes desiccation, first in the chief passages of the nose, and later in the ethmoid cells. Through the intense irritation which the dry secretion exerts upon the mucous membrane in the nose, an inflammatory process is excited and maintained in the latter. This process undergoes extension from the mucous membrane to the underlying bone, and excites in the latter rarefying osteitis. In short, ozena simplex, or chronic atrophic rhinitis, is nothing more than ulcerative ethmoiditis, resulting secondarily in atrophic rhinitis. *Goodale.*

A Cosmetic Operation of the Frontal Sinus.

RITTER (*Zeit. f. Laryn., Rhin. u. Ihre Grenz.*, Vol. V, 1912, p. 17). The operation as performed by Dr. Ritter is very similar to the Luc operation, differing, however, in certain details of technic. Ritter says that operation does not always produce universally good results as the Killian method, but sufficiently so, and the very good cosmetic result make it advisable practically in all cases, especially as when the operation is not succesful it is only required to perform a second under local anesthesia to turn it into the Killian. Ritter's method of operating is as follows: After clipping the eyebrow, an incision is made through the soft tissues down to the bone, beginning on the supraorbital ridge, just inside of a line drawn upward from the inner angle of the eye; this is carried in a curve down the side of the nose, close to the bridge, to the

upper edge of the pyriform aperture. An exploratory opening is then made in the floor of the frontal sinus, jut above the place where the frontal bone, the lacrimal bone, and the frontal process of the maxilla come together. At this point it is important to bear in mind the possible existence of a large frontoethmoidal cell. If there has not been a previous X-ray picture taken, the entering of an ethmoidal cell instead of the frontal sinus can be ascertained by passing a dull probe against the roof of the cavity opened by the exploratory incision. If the cavity is an ethmoidal cell, the probe will easily break through the roof into the frontal sinus, but the internal wall of the frontal sinus itself is so resistant that it cannot easily be penetrated. Having determined upon the size and condition of the frontal sinus, the anterior ethmoidal cells are opened up by removing the frontal process of the maxilla and the lacrimal bone. Ritter believes that the removal of the nasal bone, as advised by Luc, is not necessary. The ethmoidal cells can now be easily opened up to the extent of their involvement, and if necessary the sphenoidal sinus can likewise be opened. In clearing out the ethmoidal cells a great deal of care should be exercised not to wound the cribriform plate or to tear out bundles of the olfactory nerve. Either of these accidents may lead to intracranial complications. Ritter first opens the ethmoidal cells, leaving the median wall of the ethmoidal bone intact until all the cells have been thoroughly removed; also in using forceps he is careful to hold the blades crosswise, and in using a curette works from above downward and outward. After having removed and opened up the cells, the median wall of the ethmoidal bone is pushed gently outward by a spatula, which is introduced into the incisura olfactoria. The removal of the internal or median wall must be done under good vision, and consequently after the bleeding has been stopped, so at this point he packs the ethmoidal labyrinth and proceeds at once to the opening of the frontal sinus. In removing the inner wall of the labyrinth a good cutting conchotome should be used, and a stump left about three millimeters high. When the frontal sinus is small, sufficient room for the removal of the floor is obtained by the first incision. If it is a very large sinus the skin incision must be prolonged outward just beneath the ridge of the supraorbital margin, past the supra-

orbital notch, cutting the supraorbital nerve and vessels. Also the attachment of the cochlea must be removed from the roof of the orbit, and severed from its atachment to the periosteum covering the supraorbital margin In removing the floor of the frontal sinus there is frequently left a sharp ridge of bone running inward from the supraorbital margin. To gain a good insight into the upper part of the sinus this ridge must be removed by chiseling upward, the handle of the chisel being held close to the face. If now all portions of the frontal sinus, especially the extreme upper limit, cannot be reached by the curette, another incision is made through the skin of the forehead at a point over the upper limit of the frontal sinus, as shown by measurement with a probe. The frontal sinus is again entered at this point, all septa broken down, and the mucous membrane thoroughly removed.

The upper opening should be extended outward, according to the limits of the frontal sinus. Before closing the wound, if the cochlea has been separated from its bed, it must be brought forward and fastened by a suture to the periosteum of the supraorbital margin. If this is done chances of double vision are very small. The nasal cavity is packed with iodoform gauze for twenty-four hours. Then after a few days, if the discharge starts, the nose is merely douched with warm salt solution. Ritter says that after this operation the frontal sinus does not fill up solidly with fibrous tissue, and that, therefore, a frontal opening into the nose must be retained in order that the secretions from the granulating tissue may find an exit. He himself judges as to the patulency of the frontal sinus opening by the condition of the patient, such as headache and swelling in the upper lid. Should such symptoms develop, he examines the opening and curettes away carefully any granulation tissue, increasing the lumen of the opening until he can enter the sinus cavity easily. The next day he cauterizes with chromic acid and after that passes fairly regularly large metal bougies. If after the lapse of several months the discharge does not stop and there is other reason for interference, the frontal wall of the sinus can be easily resected under local anesthesia, converting it into a Killian operation. By the above technic Ritter has operated upon thirty cases with twenty-eight cures. *Wood.*

The Peroral Route to the Base of the Skull, the Posterior Part of the Nose and the Sphenoid Cavity.

KUHN, Kassel (*Zeitschrift für Laryngologie,* Band 4, Heft 2, p. 161). The inventor of the peroral intubation has as a result of this procedure suggested that the base of the skull, as well as the sphenoid cavity, can easily be reached by the following procedure: The patient is first anesthetized in the ordinary way. The peroral apparatus, which consists in an intubation tube attached to a flexible metal tube, is passed into the larynx. Tampons attached to threads are now packed down around the tube at the base of the tongue. The patient's head is hung over the end of the table, and a transverse incision is made at the junction of the hard and soft palates. The hamulii pterygoidei are both broken with a chisel and the bleeding easily controlled. The view to the choanæ and the base of the skull is now unobstructed and any operative procedures can easily be carried out. At the end of the operation the edges of the mucous membranes are very carefully coapted. Kuhn has already operated two cases of this character with great success. The first was a tumor the size of a hen's egg, which occupied the nasopharynx. The bleeding after removing the tumor was tremendous, but on account of the accessibility of the field easily controlled. Microscopic examination proved it to be an angioma fibrosum. The second case, a round celled sarcoma, was likewise easily and completely removed. In conclusion, Kuhn claims that this method is the logical route in attacking a tumor of the hypophysis, but as yet has had no opportunity of trying it on the living.

Horn.

The Influence of Nasal Resonance On the Singing Voice.

RETHI (*Wiener medicinische Wochenschrift,* September 7, 1912). The nose, pharynx, epiglottis and ventricular bands have a great effect on the voice. If all these parts, which are situated above the vocal cords, are cut out of a larynx that has been removed, and the cords are then brought together and air forced through the air passages from below, under proper pressure, a certain tone is produced, which varies according to the amount of the pressure.

The strength and quality of the voice depends upon the space situated above the vocal cords, i. e., the upper larynx, pharynx, mouth, nasopharynx, and nose.

In nearly every person this space is fashioned differently, particularly in respect to its size and structure. The slightest variation affects the timbre of the voice. This is even true in twins who apparently have nasal organs and throats exactly alike.

Enlarged tonsils and lingual tonsils cause decided changes in the voice which are noticeable even to the uneducated ear. Such individuals, in speaking, pronounce M as B, and N as T. The same changes in the voice are also produced by adenoids. In these cases the development of the bones of the face is interfered with by the nasal obstruction, the nose and naso-pharynx being small and narrow, the palate highly arched, etc. If these abnormal conditions are not corrected until late in life, the voice may not be favorably influenced.

Of the greatest importance is any form of nasal obstruction, such as deviations of the septum, polypi or hypertrophies of the mucous membrane.

It is self-evident that the quality of tone and resonance of the voice is to a great extent dependent on the nasal chambers, and particularly the size of the resonance chambers. We have only to think of musical instruments with resonance chambers, such as the cello, violin, and piano. The larger the resonance chamber the fuller the tone.

Singers whose noses are not clear always endeavor to improve the strength and carrying qualities of the larynx by forcing the voice. This does not make the voice sound stronger, however, but rather more muffled. It does not sound stronger in a large hall and in a small hall it sounds too sharp. Singers who have to force their voices in this way, frequently suffer a complete loss of voice and secondary inflammatory changes in the larynx. These laryngeal changes may of course be primary, and are also the result of poor methods of singing.

If in such cases in which the conditions are not caused by primary trouble in the larynx itself, but are the result of faulty methods or pathologic conditions in the nose, nasopharynx or pharynx, local treatment is used, the singer is not only not benefited, but may be injured.

On the other hand, proper operative procedures for nasal obstruction or abnormal conditions of the nasopharynx and pharynx are followed by the most satisfactory results. It is a common experience that singers who before such operations

had to force the voice in order to produce high tones, were able not only to reach their upper tones without difficulty, but increased their register. *Theisen.*

The Close Physiopathologic Relationship Between the Hypophysial System and Chronic Lesions of the Nasopharynx and the Sphenoidal Sinus.

CITELLI (*Archiv. Italiano di Otologia,* Vol. XXIII, January 1, 1912) claims that the rhinologist is the person best adapted to intervene both surgically and therapeutically on both the central and pharyngeal hypophysis.

He gives a complete statistical report of his anatomic observations of the pharyngeal hypophysis in children, which goes to show that very often the children suffering from adenoids present symptoms which are distinctly due to pressure on the hypophysis.

He has demonstrated the frequent existence in extrauterine life of an anterior portion of the hypophysis of the pharynx (called by him the vertical part), which leads to the epithelial lining of the mucous membrane, and represents a sort of external portion of the hypophysis; upon which various diseases of the nasopharynx can more readily act.

He refers to the frequent existence of the circulatory relationship between the pharyngeal hypophysis and the pharyngeal tonsil. He has had a proof of this fact by the presence of chronic congestion of the pharyngeal tonsil, the nasopharyngeal mucous membrane, and the pharyngeal hypophysis, as well as the close circulatory connections across the sphenoid between the vault of the nasopharynx and the hypophysis cerebri, and that the pharyngeal hypophysis is more superficial in children than in adults.

The gross pathologic observations of the few cadavers of infants, show that the hypophysis cerebri is more developed and weighed more in those infants who had adenoids than in those who did not at the same age. The histologic examination of the hypophysis in adenoid types and nonadenoid types of the same age was as follows:

Few eosinophiles and basophiles; connective tissue cells with large nuclei and abundant protoplasm of reddish violet color, with a tendency to collodial fusion; some nuclei devoid of protoplasm, and in some portions of the glandular lobe con-

siderable glandular tubules filled with chromophiles; considerable colloidal substance. In these cases he did not find the pharyngeal hypophysis. The sections were made at the equator, and the specimens fixed in formalin and stained with hematoxylon.

If one compares results obtained from observations of the hypophysis of nonadenoid children of the same age, it is evident that there existed from the above histologic report a hypersecretion and a hyperplasia of the glandular (adenomatous) tissue in the adenoid type.

The result of five cases shows that in all the children with adenoids he found conclusive evidence of hypersecretion and hyperplasia of the hypophysis.

The therapeutic and clinical observations go to confirm what he has already maintained, that a good many of the symptoms seen in children with adenoids are probably due to lesions of the hypophysis. The three clinical cases described by him go to show that the adenoid children present an important psychic syndrome, which can be cured by the hypophysial treatment with local treatment; that is to say, without modifying the nasal respiration, the oxygenation of the blood, or the circulatory relationship between the nasopharynx and the cerebrum. The symptoms will also disappear by operating on the adenoids, but do better by associating the hypophysial treatment with the operation.

If one adds that the same symptom has almost become pathognomonic of lesions of the hypophysis—particularly of tumors of the hypophysis without acromegaly—it is evident that you cannot help but attribute a hypophysial pathogeny to the above troubles in adenoid children. As the extract of the hypophysis can cure this syndrome, we must admit a hypofunction of that organ in adenoid cases.

The increase of secretion and hyperplasia of glandular elements which he has found in cadavers of adenoid children should be interpreted as abnormal, and by anology one sees the same histologic changes in goiter with an increase of colloidal substance, hyperplasia and hypersecretion, with, nevertheless, a hypofunction of the thyroid, which is bettered by the thyroid treatment.

The extract of the hypophysis is made from the posterior lobe, and is well tolerated by the patients, who occasionally may complain of slight palpitation, but nothing worse.

He states that in connection with the above syndrome the question of growth (infantilism and more easily excessive development of the body) that one encounters with frequency in the adenoid types, could be in part or entirely due to the disturbance of the hypophysis, given the important function that it exerts on the growth of the individual.

Other facts to uphold his theory: The influence of some tumors and other chronic lesions of the vault of the pharynx on the hypophysial system.

A barber of Catania complained of difficulty in breathing, loss of memory, drowsiness, torpid intellect, and upon examination Citelli found a large fibroid tumor in the nasopharynx, after removal of which the symptoms gradually disappeared. That besides adenoids, tumors, and chronic inflammation of the vault of the pharynx, these symptoms of hypophysial disease may be produced.

In one of his recent publications he maintains that affections of long standing of the sphenoidal sinuses (chronic suppurations, tumors, etc.) can influence the hypophysial system on account of the intimate relationship of the sphenoidal sinuses to the sella turcica; which he upholds by anatomic and clinical facts.

Citelli concludes that: 1. His researches of normal anatomy have demonstrated that there exists an intimate relationship between the pharyngeal tonsil and the vault of the nasopharynx on the one side, and the hypophysial system on the other side.

2. His researches on the histology and pathology have also demonstrated that in the adenoid types the central hypophysis often presents a hypersecretion and hyperplasia of the glandular elements.

3. He has described an important psychic syndrome in the adenoid children; a syndrome which consists of well known aprosexia, and which can disappear alone with hypophysial treatment. This syndrome is identical with the one that is encountered in the hypophysial affections, and was equally demonstrated in a case of soft fibroma of the vault of the pharynx (the removal of the tumor was followed by a cure). Some of the phenomena of this important syndrome have also been observed in many cases of hard fibroma of the nasopharynx.

4. His theory is sustained by so many facts and consider-

ations, that it proves that there is a constant congenital predisposition or acquired condition. The influence of possible diseases of the vault of the pharynx and the sphenoidal sinuses on the pathology of the hypophysis, cannot seem more improbable than the origin of certain hypophysial symptoms or group of symptoms, which may appear in the above affections.

5. His theory, which admits of a relationship between the hypophysial system and adenoid vegetation, is not to be confused with that of Poppi's, which is founded on the hypotheses and personal observations of other authors. This latter theory admits of an embryonal lesion of the base of the cranium, and gives rise to alterations of a primitive embryonal nature of the central hypophysis. This alteration of the central hypophysis would cause a lesion of the epiphysis, and the relationship between the hypophysis and the thyroid and thymus glands would be the cause of the lymphatism and consequently of the adenoid vegetation.

6. His theory, which is based on numerous facts, admits that the hypophysial lesions can be provoked (in predisposed individuals) by adenoid vegetation and other affections of the vault of the pharynx and the sphenoidal sinuses.

Lucchetti.

III.—LARYNX.

Multiple Amyloid Tumors of the Larynx and Pharynx.

PAUL SECKEL (*Archiv. für Laryngologie und Rhinologie*, Bd. 26, Heft 1). A man 62 years old, with a clinical diagnosis of thrombophlebitis, multiple emboli and cardiac weakness, showed at the autopsy a condition simulating chronic verucous tuberculosis of the larynx and pharynx. Examination showed these growths to consist of amyloid substance. Chronic tuberculous peritonitis and tuberculosis of the lungs and kidneys were present. *Goodale.*

Leiomyoma of the Larynx.

DONOGANY (*Monats. f. Ohren. u. Laryngo-Rhinologie*, Vol. XLVI, 1912, p. 540). A boy eleven years old began to become hoarse about seven months before his examination, and for the last fourteen days has showed marked evidences of dyspnea. On examination a smooth tumor about the size of a hazel nut, with surface and color about the same as surrounding region, was seen, filling almost the whole of the

laryngeal orifice. Tracheotomy was done and the tumor then removed endolaryngeally. The growth sprang from the left ventricular band and the anterior commissure. Histologic examination of the throat showed it to be a leiomyoma.

Wood.

Recurrent Paralysis Caused by Stenosis of the Ostii Venosi Sinistri.

B. Freystadtl and J. Stranz. (*Monats. f. Ohren. u. Laryngo-Rhinologie*, Vol. XLVI, 1912, p. 557). In the case reported the postmortem showed that as the result of a mitral stenosis there was a very greatly dilated left auricle which pressed upon the left recurrent nerve sufficiently to produce paralysis. As the postmortem was a complete one, it was easily shown by a dissection of the nerve that there could be no other cause for this paralysis. The musculature of the left side of the larynx was badly atrophied, and histologically the thyreoarytenoideus internus and externus showed degenerative changes; histologic examination of the recurrent laryngeal nerve likewise showed marked degeneration. *Wood.*

A Malignant Tumor of the Larynx Undergoing Spontaneous Healing.

Broeckaert (*Zeit. f. Laryn., Rhin. u. Ihre Grenz.*, Vol. V, 1912, p. 51). The patient, fifty years of age, presented himself with a tumor which blocked pretty near the whole lumen of the larynx so that tracheotomy at first seemed necessary. The patient, after his entrance into the hospital, became gradually better, and the growth came away in several large ill-smelling necrotic pieces which were rather easily removed with forceps. The wounded surface healed smoothly, and in a few days the patient was well without any sign of a tumor in the larynx. The patient, however, came back after a few months with a slight recurrence on the left vocal cord near the anterior commissure. A portion of this was removed and examined histologically. The growth proved to be of a very rare type, being a hyalin adenocarcinoma. *Wood.*

Concerning the Diagnosis of Carcinoma of the Larynx.

Fein (*Monats. f. Ohren. u. Laryngo-Rhinologie*, Vol. XLVI, 1912, p. 69) reports an interesting case of carcinoma of the larynx which he had observed over a period of eight and one-half years, the patient having had trouble with his throat for

six years before he came under Fein's care. No radical operation was attempted, but from time to time bits of tumor mass were removed and studied histologically. He finally died from an extensive involvement of the larynx and surrounding portions of the pharynx. Sometimes the clinical features presented during the course of observation were such as to make a diagnosis of malignancy practically impossible by clinical measures. The interesting feature of the case, however, was that from the first the pathologic diagnosis was always carcinoma, and that this diagnosis was made by the pathologist independent of any clinical data. Because of this fact Fein believes that there should always be an accurate histologic study of portions of bits of the tumor mass in suspicious cases, and also that the pathologist should make his diagnosis independent of any clinical data. *Wood.*

The Ambulatory Treatment of Laryngeal Tuberculosis.

MEYER (*Zeit. f. Laryn., Rhin. u. Ihre Grenz.*, Vol. V, 1912, p. 35) in this article goes over rather thoroughly all the various methods of treating cases of laryngeal tuberculosis while they are still able to attend to their work, but there are several rather interesting features worth noting. He is a strong believer in the use of tuberculin, except in cases with marked lung involvement, high fever, intestinal complications, cachexia or an inclination toward hemoptysis. Following its use, usually in the larynx, there is hyperemia with slight desquamation of the tissues, and in one case there was a slight whitish transparent membrane; never any edema or acute spreading of the tuberculous process. In connection with the general treatment active local therapy is always advisable, except when the general condition of the patient is very bad or the disease in the larynx is extensive or progressing beyond the limits of the larynx to the throat. Acute miliary process in the larynx and extensive involvement of the trachea also contraindicate any active local treatment. He is very favorable to the use of the electric cautery, believing that not only superficial ulcers may be easily destroyed, but that also the cautery works beyond the area of absolute destruction, due to a reactive inflammation in the surrounding regions. He believes that much can be accomplished by absolute rest of the voice, but that whispering does not do this, and the patient should be instructed to avoid speak-

ing. He says that the larynx constricts during the attempt to whisper. He also calls attention to the use of the alcohol injection into the superior laryngeal nerve for the purposes of relieving dysphagia, though he does not believe that this method of treating has much effect as far as cure is concerned.

Wood.

Stenosis of the Bronchi Caused by Dilatation of the Auricle.

KAHLER (*Monats. f. Ohren. u. Laryngo-Rhinologie,* Vol. XLVI, 1912, p. 553) examined eleven cases of mitral stenosis, one case of pure mitral insufficiency, and one case of aortic stenosis. The reported changes were all observed through the bronchoscope. While it is well known that the bifurcation carina of the trachea in the large majority of cases occupies a position somewhat to the left of the median line, Kahler found that in his patients, ten times it occupied a position almost in the middle, once to the left, and twice to the right. In the case in which the carina was placed somewhat to the left there was very little enlargement of the auricle. In three of the patients with marked enlargement of the auricle there was a sharp bending of the left bronchus, making almost a complete right angle. In all of the cases with dilatation of the auricle the left bronchus was found more or less constricted, in some the stenosis being very marked. Also the direction of the stenosis seemed to run parallel with the enlargement of the auricle. In two cases with marked enlargement of the right heart the right bronchus was somewhat flattened. In the most of the cases the mucous membrane in the region of the stenosis was congested, possibly as a result of the existing bronchitis. The enlargement of the auricle, as compared with the pulsation at the bifurcation, caused the carina to move from the left to the right, due to closer contact of the bifurcation with the aorta than is usually found. Also there was almost always a pulsatory uplifting of the flattened portion of the lower anterior part of the left bronchus. In three cases the enlargement of the auricle produced a left sided paralysis of the recurrent laryngeal nerve.

Wood.

The Treatment of Laryngeal Tuberculosis with the Special Consideration of the Dysphagia.

AURELIUS RETHI (*Monats. f. Ohren. u. Laryngo-Rhinologie,* Vol. XLVI, 1912, p. 910) reports a case in which, three weeks

after an injury, there developed tuberculosis of the larynx at the seat of the trauma. In this case there was no evidence of pulmonary infection, and he believes that the infection was carried through the blood. Concerning dysphagia, he says that the pain is probably due to a hypersensitiveness of the nerves, and that this hypersensitiveness is induced by a continuous irritation such as we would find in ulceration; or else by the pressure of granulation tissue on the nerve endings, as in simple infiltration. As a demonstration of this hypersensitiveness, he claims that pressure over the entrance point of the superior laryngeal nerve into the larynx will cause a tickling sensation in the throat with cough. It is also interesting to note that the pain in these cases is temporarily relieved and the patient can for a short time swallow easily if rather severe pressure is put on the hypersensitive nerve. Concerning the relief of the dysphagia, Rethi says that the sensibility of the superior laryngeal may be destroyed by cutting the nerve or by the injection of some chemical material. When the disease involves the upper or anterior surface of the epiglottis, these procedures have no effect, as this part of the larynx is supplied by the glossopharyngeal. He reports two cases of severe dysphagia in which he did a double resection of the superior laryngeal nerve. Before the operation the sensibility of the larynx was normal; afterwards, in both cases, the posterior surface of the epiglottis, the aryepiglottic fold, and the ventricular band were anesthetic, while the sensitiveness of the vocal cords was only partially lessened. This probably was due to the anastomosis of the internal branch of the superior laryngeal nerve with the recurrent nerve. *Wood.*

IV.—MISCELLANEOUS.

Researches on the Bacteremia in Oto-Rhino-Laryngology.

C. CALDERA (Proceedings of the 14th Italian Congress of Oto-Rhino-Laryngology, Rome, October 26-28, 1911,) in a series of researches, undertaken with a view to establishing the sterile condition of the blood in patients suffering from acute suppurating diseases of the ear, nose, and throat, concludes that in normal conditions, that is to say, where there exists no symptoms of general pyemic reaction, there is never any concomitant bacteremia in the nose and throat affections; simple

bacteremia is encountered, on the contrary, quite often in lesions of the ear, and is, therefore, not of itself such an unfavorable prognostic sign. He explains the above on anatomic grounds. *Lucchetti.*

On the Question of a Laryngeal Center in the Cerebral Cortex.

GRABOWER (*Archiv. für Laryngologie und Rhinologie,* Bd. 26, Heft 1). The conclusion is drawn that the cortical area described by Rothman and Katzenstein in the anterior lobule of the cerebellum is not the place which contains a cerebellar laryngeal center. It is, however, probable that such a coordination center exists somewhere in the cerebellum.

Goodale.

The Lymph Channels of the Upper Nasal Region and Their Relation to the Perimeningeal Lymph Spaces.

ZEWILLINGER (*Archiv. für Laryngologie und Rhinologie,* Bd. 26, Heft 1) reaches the following conclusions: 1. The anatomic proof of the relation of the lymph channels of the upper nasal territory with the lymph spaces of the central nervous system is established in man. 2. It is established that there is present a superficial lymph net in the upper region of the nasal chambers, independent of the perineural lymph channels of the olfactorious. This network, and also the above mentioned perineural lymph channels, are connected with the perimeningeal lymph channels. The ways in which the postoperative and other meningeal and cerebral complications occur from the nasal chambers are, in addition to the blood current, the lymph channels of the upper division of the nasal passages, which stand in direct relation with the perimeningeal lymph spaces. *Goodale.*

Congenital Fistulæ of the Neck.

LEEGARD (*Archiv. für Laryngologie und Rhinologie,* Bd. 26, Heft 1) reviews the various types in a long and comprehensive paper and comes to the following conclusions:

Lateral Fistulæ. I. The preponderating frequency on the right side is probably explained by the normal history of development. II. In view of the cases the external opening of the fistula lies below the clavicle. III. The situation of the inner opening appears more constant than has hitherto been assumed, in that it almost always lies near the margin of the

posterior pillar of the palate. IV. A single lumen only is present, at least exceptions are extremely rare. V. The epithelium of the fistulæ shows no regular arrangement. Pavement and cylindrical epithelium are found throughout the whole length. VI. A lymphoid layer was found in the cases examined at almost every level. It can therefore be assumed that they had their origin from the entodem in nearly their whole length. VII. The entoderm plays consequently the chief role in the formation of the fistulæ even when the fistulæ is externally incomplete. VIII. If the fistula is elevated by deglutition, this symptom indicates that a complete fistula is present. IX. Those fistulæ which are incomplete externally have often a much greater extension inwards than one can establish by clinical examination. An apparently solid strand leading to the pharynx must consequently be regarded as a fistulous passage, and treated at the time of operation. X. The injection treatment with caustics should not be entirely given up. XI. Operative treatment with simple extirpation is preferable.

Median Fistulæ.—Besides those arising from the thyroglossal duct, there is another group of median fistulæ, where it must be assumed that they have another history of development. *Goodale.*

Angioma of the Pharynx, Larynx, and Neck.

FALLAS, Brussels (*Archives Internationales de Laryngologie, D'Otologie et de Rhinologie,* January and February, 1912). In angioma the essential element consists in the new formation of vessels with a small quantity of connective tissue. We also meet with tumors formed by dilatation of preexisting vessels. There are four kinds of angioma—arterial, venous, capillary, and cavernous.

Arterial angioma are diffused tumors without limited borders, with an uneven surface, giving the impression of macaroni, covered by the skin. The color is a bright red, and they do not give rise to pulsation. Venous angioma are formed by veins with thick walls which often anastomose. The color is a bluish red, and without pulsation. Capillary angioma appear as winelike blotches so often seen on the skin. Cavernous angioma are formed of large irregular cavities limited by layers of connective tissue, containing smooth muscular fibers arranged without order. The blood sacs are lined throughout

their extent with an endothelial covering. Their limits are often very diffuse. Sometimes, however, they have a capsule which extends along the afferent and efferent vessels. The author reports a case which belongs to this form of angioma. The patient was a girl aged 19, who consulted him because of a certain roughness of her voice. This was aggravated at the time of menstruation and whenever she became angry. In childhood she had been operated upon in the left side of the neck for a large bluish tumor, similar to a small mass still showing on the left surface of the larynx. On the anterior surface of the neck, slightly to the left of the median line, above the manubrium, several venous dilatations were to be seen of a moderate size with similar characteristics. The posterior surface of the pharynx was covered by an irregular mass of dilated vessels of bluish color, resembling veins, and which continued upward toward the epipharynx, and downward toward the larynx, as blood vessels of more normal aspect. The arytenoids were covered by a bluish mass which partly obscured the larynx. The same picture was to be seen upon the false cords, the left vocal cord, and even extending into the subglottic region. The patient otherwise had no complaint; there was no dyspnea, no cough, she had never had any hemorrhage. Inasmuch as there were no serious symptoms complained of, and because of the great multiplicity of the angioma, no interference was advised.

These tumors, according to the author, can develop as the result of trauma, but generally they are congenital and often form malformations rather than tumors, properly speaking. They are found more frequently in girls than in boys. They are met with in all parts of the body, but especially upon the face and neck. This latter effect is due, according to Virchow, to defective formation in the facial and branchial valves. This genesis explains why at the same time they are cutaneous and subcutaneous. Angioma are often deep and extend even to the subjacent mucosa. Angioma of the larynx, however, are not frequent. According to Lenox Browne, they are the rarest of all benign tumors of that organ. Fauvell has reported two out of 300 cases of benign neoplasm. Frequently they cause no trouble and are only accidentally discovered. At other times trouble with phonation calls attention to them, as in the author's case. In other cases there may be attacks

of coughing and even dyspnea. Difficulty with swallowing is rare. The prognosis in the case of angioma is variable. Venous and capillary angioma are generally stationary, but they can increase in size under the effects of trauma, the menstrual period, etc. Arterial angioma are characterized by the tendency to increase in size, and can become dangerous by their size. They are not infectious, but can cause local troubles, such as an erosion of the underlying bone. Angioma, furthermore, can become inflamed, ulcerating and giving rise to severe hemorrhage at times. The greatest danger arises from operation with cutting instruments. Ferreri has reported a case where he removed such a growth in a man aged 60, and where, on account of the profuse hemorrhage, it was necessary to do a tracheotomy and tampon the larynx. The patient died of pneumonia forty hours later. In place of cutting instruments, the hot wire or the galvanocautery are to be recommended. Injections into the tumor of hemostatic liquids, such as tannin, chlorate of iron, and alcohol, have been used with greater or less success. Malhou has used injections of oxygenated air with success. Electrolysis has also given brilliant results. Finally, radium has been employed in recent years with particularly satisfactory result. *Harris.*

BOOK REVIEWS.

The Index of Oto-Laryngology.

JOSEPH C. BECK, M. D., Editor. Volume I, 1911. D. Winternitz, Publisher, Chicago, Ill.

It gives us great pleasure to call attention to the first bound volume of the Index of Oto-Laryngology, as well as to the current volume. Dr. Beck is furnishing to the otologist and laryngologist a veritable index medicus of the specialty. Every physician who is at all interested in the literature of the specialty should be a subscriber, and to those who do any writing this journal will be found to be indispensable, since all the articles as they appear in the literature, both English and foreign, are catalogued and the very important ones abstracted. In addition, notes of the proceedings of all the special societies are given.

This work is a labor of love on Dr. Beck's part, and we urge its support by the otolaryngologic profession. Subscriptions may be sent direct to Dr. Joseph C. Beck, Chicago, Ill.

Richards.

"Handbuch der Speziellen Chirurgie des Ohres und der Oberen Luftwege."

Volume III, Sections 3 and 4. Published by Kurt Kabitzsch, Wurzburg. Price, unbound, 14 marks.

The fourteenth separate section of this magnificent work is a volume of 450 pages and takes up the subject of intranasal surgery and diseases of the nose. Dr. Katz, the author of this part, although not connected with a university clinic, is well known in Germany as a skillful technician and as a just analyzer of modern literature.

Evidently the epoch of the "gun-shot specialist" is not past in Germany, for he says: "Diese Zeit der chirurischen Klein-

kramerei und Polypragmasie" is still with them; but he claims that rhinology is a field for the man with general surgical training, and that attention to surgical indications, surgical asepsis and surgical pathology is as necessary here as in any other branch of surgical work.

The chapter on methods of treatment is interesting. Among other things he claims that the only indication for a nasal douche is in removing the crusts in ozena, otherwise, even in skillful hands, the danger of infecting the ears is far too great; a point on which many men will differ with him. Douching the nose in babies and small children is never allowable.

One looks with interest to the chapter on the submucous resection of the septum. He thinks we should give to Krieg the credit for the window resection of the septum, and considers the later additions of Bönninghaus, Killian and others with the retention of both mucous membrane flaps as details.

The operation should be known as Krieg's window resection, and not as the submucous septum resection.

He remarks that "some American specialists have prophesied the giving up of this important operation, principally because Freer of Chicago has developed a relatively simple operation into one of such complexity that it is out of all proportion to the pathologic conditions present," a criticism which is absolutely unjust and unfair.

The chapter on reflex neuroses is short, and gives but a superficial view of this now very important branch of rhinologic pathology.

The work of Sluder on sphenopalatine-ganglion neurosis is not even mentioned, most of the weight being given to Killian's work on neuroses of the anterior ethmoidal nerve.

The etiology of ozena is thoroughly discussed, but Katz finds that, in spite of the recent bacteriologic investigation of Perez, Lautmann and others, we are as far from the truth as ever. That there is a certain hereditary predisposition he thinks there is no doubt, and it may be that the final solution

of the mystery will be brought about by a combination of several theories. The luetic basis is certainly not the proper one, as has been proved by several competent observers. When he comes to speak of the treatment, he finds nothing new, with the possible exception of the paraffin injections and the very careful exclusion of all sources of accessory cavity infections by proper surgical measures.

Taking the contribution of Katz as a whole, it cannot be considered as a text book because of its great lack of detail, but as an analysis of modern German contributions it is fair enough. It would seem that a more extensive knowledge of the American literature, by men who undertake tasks of this sort, would result in a fairer distribution of credit. As usual, the plates, especially on the pathology of the intranasal neoplasms, leave nothing to be desired. *Horn.*

CORRECTION.

Dr. Freudenthal writes to the Editor that in his paper on "Second Report on the Therapeutic Value of Radium in Malignant Tumors of the Upper Air Tract," ANNALS, March, 1912, page 337, he used the following words:

"Case 14. Doctor ——, age 70, a well-known colleague * * *. When I first saw him * * * he handed me a typewritten report of his microscopic diagnosis with the words malignant carcinoma underlined. The gentleman who had made the microscopic diagnosis is a colleague whose character and fine tact are just as admirable as his ability, and yet he considered it correct to send such a report directly to the patient."

Dr. Freudenthal writes that he has since learned that the letter containing the diagnosis was given to the patient by a member of his family, without the knowledge or consent of Dr. Jonathan Wright, who was the pathologist in question.

Dr. Freudenthal feels that he should make this correction and this public expression of regret and apology to Dr. Wright, whom he holds in the highest esteem.